Poster #1a

POSITRON EMISSION TOMOGRAPHY ABSOLUTE STRESS MYOCARDIAL BLOOD FLOW FOR RISK STRATIFICATION IN NONISCHEMIC CARDIOMYOPATHY

Thomas G. Middour, Todd M. Rosenthal, Freddy M. Abi-Samra, Michael L. Bernard, Sammy Khatib, Glenn M. Polin, Paul A. Rogers, Robert M. Bober, Daniel P. Morin
Presenter: Thomas Middour, MD, Ochsner Health System

Background: Sudden cardiac death is a substantial cause of mortality in patients with cardiomyopathy, but evidence supporting implantable cardioverter defibrillator (ICD) implantation is less robust in those with nonischemic cardiomyopathy (NICM). Absolute quantification of stress myocardial blood flow (sMBF) measured by positron emission tomography (PET) may predict adverse cardiac events.

Objective: We assessed whether sMBF predicts future ventricular arrhythmias (VA) and/or death in patients with NICM.

Methods: We prospectively followed patients with NICM (LVEF≤35%) and an ICD who underwent cardiac PET stress imaging with sMBF quantification. NICM was defined as absence of angiographic obstructive coronary stenosis, significant relative perfusion defects on imaging, and prior coronary revascularization or acute coronary syndrome. Endpoints were appropriate device therapy (VA) and all-cause mortality. Subgroup analysis was performed in patients who had no prior history of VA (i.e., the primary prevention population).

Results: Patients (60±14 years, 46% male) were followed for 41±23 months. sMBF predicted VA both in the whole population (HR per 1 mL/g/min:0.17, P=0.015) and in the primary prevention population (HR 0.13, P=0.049). Patients with sMBF below the median had significantly more VA than those above the median, both in the whole population (P=0.004) and in the primary prevention population (P=0.046). Estimated 3-year VA rates were 67% vs. 13% and 39% vs. 8%, respectively. sMBF did not predict death.

Conclusion: In patients with NICM, lower sMBF as measured by cardiac PET predicts VA. This relationship may be useful for risk stratification and decision making regarding ICD implantation.

Poster #1b

RELATIONSHIPS BETWEEN MYOCARDIAL SCAR ON CARDIAC MAGNETIC RESONANCE IMAGING AND ELECTROCARDIOGRAPHIC VENTRICULAR DEPOLARIZATION AND REPOLARIZATION

Abdulaziz Joury MD, Peter Pantlin BS, Ronald Chong-Yik MD MPH, Sangeeta B. Shah MD, Joel Xue PhD and Daniel P. Morin MD MPH FHRS
Presenter: Abdulaziz Joury MD, Ochsner Health System

Background: ECG markers of ventricular depolarization and repolarization (D/R; e.g., QRS duration, QT, Tpeak-Tend [Tpe]) correlate with risk of sudden cardiac arrest. Similarly, myocardial scar on cardiac magnetic resonance (CMR) imaging correlates with ventricular arrhythmias. Whether D/R markers correlate with scar burden is unknown.

Objective: To identify the relationship between ventricular D/R and left ventricular (LV) scar burden on CMR.

Methods: Patients without coronary artery disease (CAD) (i.e., no MI history, stent placement, CABG, or significant stenosis in epicardial vessels) underwent CMR with analysis of LV myocardial scar via late gadolinium enhancement (LGE). ECGs done within 3 months of CMR were automatically analyzed and manually overread. We assessed the correlation between D/R markers and scar burden on CMR.

Results: There were 35 patients (49% M, 41±20 Y, LVEF 56±12%). Indications for CMR were to rule out: ARVC (16), infiltrative cardiomyopathy (6), hypertrophic cardiomyopathy (5), myocarditis (4) or other (4). No patient had positive CMR for the diagnosis in question. The mean QRSd was 92±15 ms, Tpe was 104±16 ms, and QT was 399±39 ms. Mean LVEF:56%.
There was positive correlation between total volume of LV scar and Tpe (r=0.4, p=0.013) and QT (r=0.42, p=0.012), and between relative scar volume and Tpe (r=0.51, p=0.002) and QT (r=0.39, p=0.022). However, there was no correlation between total volume of LV scar and Tpec (r=0.03, p= 0.86), QTc (r=0.26, p=0.13), QRS duration (r=0.24, p=0.16), or LVEF (r=0.13, p=0.47). Similarly, no correlation was found between relative scar volume and Tpec (r=0.011, p=0.95), QTc (r=0.26, p=0.13), QRS duration (r=0.18, p=0.29), or LVEF (r=0.07, p=0.68).

Conclusion: In patients without CAD, scar on CMR correlates with the ECG repolarization indices Tpe and QT, but not with Tpec, QTc, QRSd or LVEF.

Poster #1c

A SYSTEMATIC REVIEW OF PHARMACOLOGICAL INTERVENTIONS FOR PREVENTION OF CHEMOTHERAPY INDUCED CARDIOMYOPATHY PAIRWISE AND NETWORK META-ANALYSIS

Magdy Hanna, MD, Usman Mustafa, MD, Khagendra Dahal, MD, Hussam Watti, MD, George Mina, MD, Parinita Dherange, MD, Nachiket Apte, MD and Paari Dominic#, MD.
Presenter: Magdy Hanna MD, LSU Health Shreveport

Background: Chemotherapy induced cardiomyopathy (CCMP) is a common adverse effect of antineoplastic drugs.

Objective: This meta-analysis aims to study the impact of cardioprotective drugs including statins, angiotensin converting enzyme inhibitors (ACEi)/ angiotensin receptor blockers (ARBs), beta-blockers (BB), Spironolactone and chelating agents like Dexrazoxane on left ventricular function and all-cause mortality when used as prophylaxis against cardiotoxic chemotherapy.

Methods: We searched PubMed, EMBASE and Cochrane Central from inception to December 2017. Studies evaluating the impact of cardioprotective drugs on left ventricular function and all-cause mortality in patients receiving cardiotoxic chemotherapy were selected.

Results: A total of 27 studies were included. Conventional cardioprotective drugs (CCD) significantly reduced all-cause mortality compared to placebo (OR: 0.456, 95% CI: 0.211 to 0.987, p=0.046). Dexrazoxane showed no survival benefit in patients receiving cardiotoxic chemotherapy (OR: 0.954, 95% CI: 0.519 to 1.754, p=0.880). Dexrazoxane resulted in significantly lower risk of heart failure (HF) (OR: 0.274; 95% CI: 0.206 to 0.365, P

Conclusion: Our meta-analysis suggests that Dexrazoxane decreases the risk of cardiotoxicity but does not offer any mortality benefit, while CCD not only prevents cardiotoxicity but also provides additional mortality benefit.

Poster #2a

MIND OVER MATTER: ASSOCIATIONS BETWEEN PAIN AND CARDIORESPIRATORY FITNESS

Sergey Kachur, MD, Rebecca Morera, MD, Alban Deschutter, MD, Carl J Lavie, MD, Richard Milani, MD
Presenter: Sergey Kachur MD, Ochsner Health System

Background: Even though the experience of pain is subjective and difficult to observe directly, it has a myriad of long-lasting effects on physical and mental health. We have found pain to be a significant predictor of changes in mortality after cardiac rehabilitation (CR), here we examine the interactions of reported pain and changes in cardiorespiratory fitness (CRF) as a possible mechanism of mortality effects.

Objective: Determine whether pain is a significant factor in CRF changes during CR.

Methods: 1110 subjects with stable CHD referred for CR between 01/2000 and 06/2013 with a mean follow-up of 6.3 years were examined with respect to reported pain levels using the Short-Form 36-health questionnaire before initiating CR. Increases in CRF were examined with respect to reported pain levels using receiver operating curve analyses. Using the resultant threshold, logistic regression was performed to assess adjusted associations between pain and increases in CRF.

Results: Pain levels were effective at discriminating between those with increased and unchanged/decreased CRF (Figure 1) with an area under curve of 0.53. Multivariate logistic regression showed that low pain was inversely associated with increases in CRF (OR 1.68, p=0.01). In introducing pain as an adjusted predictor of mortality, depression was no longer a significant predictor of CRF response during CR.

Conclusion: Pain is inversely related to increases in CRF during CR. Pain also appears to be a confounding factor for changes in CRF previously attributed to depression. Lowering CRF response may be one mechanism in which pain affects mortality. Further study is needed to determine if modifying pain can improve CR outcomes.

Poster #2b

OUTCOMES OF CHRONIC RESYNCHRONIZATION THERAPY (CRT) WITH OR WITHOUT A DEFIBRILLATOR IN CHRONIC HEART FAILURE PATIENTS - AN UPDATED SYSTEMATIC REVIEW AND META-ANALYSIS.

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Presenter: Parinita Dherange MD, LSU Health Shreveport

Background: Chronic Resynchronization Therapy (CRT) reduces morbidity and mortality in selected patients with systolic heart failure. In addition, it also reduces ventricular arrhythmias, making the choice between CRT-D (CRT – Defibrillator) and CRT-P (CRT – Pacemaker) contentious despite a significant difference in cost involved in implantation.

Objective: To assess the risk of all-cause mortality and sudden cardiac death (SCD) in patients receiving CRT with or without a defibrillator.

Methods: We performed a literature search from inception to 26 August 2018 to obtain all studies that evaluated risk of all-cause mortality and/or SCD among patients receiving CRT-D versus CRT-P. Data was pooled using a random-effect model. Event rates were used as a measurement of treatment effect.

Results: A total of 33 studies with 89,668 patients (mean age of 68.3 ± 5.4 years, QRS interval of 159 ± 8 msec, Left ventricular ejection fraction of 25 ± 2% and 49% had ischemic cardiomyopathy) that compared CRT-D (74,877) Vs CRT-P (14,791) were included. The mean follow-up period was 2.8 ± 1 years and majority of the patients were males. The pooled data of 31 studies, which assessed all-cause mortality as an outcome revealed that CRT-D patients had significantly lower all-cause mortality than the CRT-P patients (OR: 0.777, 95% CI: 0.673 to 0.896, P=0.001, I2=58) (Fig 1). Twelve studies that assessed SCD as an outcome revealed that CRT-D patients had significantly lower SCD than the CRT-P patients (OR: 0.453, 95% CI: 0.271 to 0.758, P=0.003, I2=0) (Fig 1). Univariate Meta-regression showed significant interaction between diabetes (details available in 16 studies) and all-cause mortality in patients receiving CRT explained by smaller differences in outcome between CRT-D and CRT-P groups with increasing diabetic population in the study.

Conclusion: Overall, there is a significant survival benefit in terms of all cause mortality and SCD by adding defibrillator to CRT patients.

Poster #2c

QUALITY CARE ANALYSIS OF THE APPROPRIATENESS OF VA CARDIOLOGY CLINIC VISITS

Ayush Motwani, MD; Twinkle Singh, MD; Karnika Ayinapudi, MD; Mark Cassidy, MD; Anand Irimpen, MD
Presenter: Ayush Motwani MD, Tulane School of Medicine

Background: Southeast Louisiana Veterans Health Care System (SLVHCS) provides quality, compassionate, safe health care to Veterans throughout 23 parishes in southeast Louisiana. Specialty care is available through the New Orleans and Baton Rouge clinics and inpatient services are coordinated through community facilities and other Veterans Affairs Medical Centers. New Orleans VA Cardiology Clinic was cited in 2016 for highest number of cardiology clinic appointments in unit time across all specialties and VA practices across the country. There were questions raised, if this huge volume of patient encounters is due to sicker population in the area, lack of other nearby VA facilities, lack of primary care availability or due to unnecessary close follow up visits from physicians. On our initial evaluation during the time period of august 2016, we found that excess visits were mainly secondary to unmandated close follow up visits. In order to decrease this our interventions included conferences to discuss appropriate follow up and distribution of flyers in each clinic room with criteria for appropriate follow up time interval and discharge. Our current study was designed to see if there was a decrease in the number of unnecessary visits following these interventions.

Objective: 1.To assess whether the follow up of patients in clinic was appropriate in terms of need for follow up and appropriate time interval of follow up.
2.If the results of the initial analysis impacted appropriate decision making in terms of follow up of patients.

Methods: A team consisting of 3 fellows, clinic nurse, supervising physicians was formed. We identified 220 patient encounters in the cardiology clinic during a one-week period from April 16 2018 to April 20, 2018.The visit type and sub-specialty was determined Nurse practioner visits and sub-specialty visits (CHF, Vascular, EP) were excluded from further detailed chart review. Final chart review was done on 99 general cardiology visits seen by physicians. The main parameters assessed included the reason for each visit, timing of last cardiology visit (within 1, 2, 3, 6, 9, 12, 24 months or >24 months), timing of next follow up cardiology visit (within 1, 2, 3, 6, 9 or 12 months or prn or if discharged from clinic and the reason for follow up visit. Current data was compared to previous data as regards to time interval to follow up and appropriateness of follow up. As mentioned previously, predefined criteria for follow up were used to determine if the time interval of follow up was appropriate or not. These criteria of time duration to follow up were mainly determined based on urgency of follow up, symptomatology, co-morbidities, medication changes, and follow up of results of specific cardiac testing.

Results: Out of the total 220 patient visits, 57.8% were general cardiology visits (NP and MD visits) while the total number of general cardiology visits seen by MDs alone were 99 encounters. 8% of patients were asked to follow up within a month compared to 21% on

the previous analysis (p=0.0082). 26% patients were asked to follow up within 3 months compared to 47% before (p=0.0016). 22% were asked to follow up within 6 months compared to 17% and 11% were asked to follow up at 12 months compared to 4% before. 2% of patients were discharged from the clinic. 53% of these visits were stable CAD patients and 15% of these were stable atrial fibrillation patients. Remaining were hypertension. hyperlipidemia, unexplained dyspnea, valvular heart disease, congestive heart failure. 47% of the visits could still be termed as inappropriate follow up mostly as regards to the time duration with early follow up.

Conclusion: The interventions that were instituted resulted in a decrease in follow up visits at 1 month or 3 months. Further interventions may be beneficial to further decrease follow ups. These could include reiteration of criteria of follow up and discharge in clinics and also continuing physician education. Designating team leaders in each clinic group for assessment of quality and quantity of visits could also make a difference. A decrease in such number of unnecessary follow ups through appropriate interventions may provide more opportunities to see new patients leading to more access to healthcare facilities.

Poster #2d

THE LONG-TERM CARE OF ADULT CONGENITAL HEART DISEASE PATIENTS: A SURVEY-BASED STUDY

Danielle Hatt, MSIV; Brittany Ballew, MSIII; Natasha Lalos, MSIV; Dr. Thomas Young, MD; Dr. Diego Lara, MD
Presenter: Danielle Hatt MSIV, Ochsner Health System

Background: Over one million adults in the United States are estimated to have congenital heart disease (CHD). This is due to advances in medical knowledge and technology, producing a unique population of patients requiring specialized and multidisciplinary care. Many adults living with CHD (ACHD) struggle to establish optimal care for their condition. This is because the number of specialist centers and practitioners is minimal. Formal ACHD board certification was established in 2015.

Objective: The aim of this study was to identify disparities in long-term health outcomes of ACHD, comparing those who see cardiologists who are not ACHD board-certified vs. ACHD board-certified physicians.

Methods: ACHD answered a standardized questionnaire, reporting on their current cardiac medical care. Participation was elicited through social media conducted by the Adult Congenital Heart Association (ACHA). Analysis included intra-cardiac condition comparison to identify generalizable disparities, along with those that may be condition-specific. Thematic analysis was utilized for the qualitative arm of the study.

Results: 245 ACHD patients completed the survey. Respondents hailed mostly from Texas, California, and Pennsylvania (10.2%, 9.8%, and 8%, respectively). 75% of respondents reported seeing an ACHD-certified physician. Most respondents see a local cardiologist, while 20% traveled over 100 miles, regardless of their physician's training. Frequency of follow-up did not differ based on physician certification. Those seeing an ACHD-certified physician reported more medications and more cardiac-related hospitalizations and surgeries than their counterparts.

Conclusion: ACHD board certification is a developing practice. States with large response rates have more speciality clinics and patient self-advocacy. There is support for further elucidation of these states' health promotion strategies for replication in more underserved areas. It has been postulated that long distance to an ACHD-certified physician may be a deterrent; our data did not support this, and it would be worth future efforts to inform patients of their proximity to an ACHD-certified physician. The qualitative survey responses reflected inconsistent knowledge regarding the certification of their cardiologist, highlighting that patient education is not yet optimum to embrace the growing field.

Poster #3a

ARRHYTHMIAS IN HEMATOPOIETIC STEM CELL TRANSPLANT RECIPIENTS: SHOULD WE WORRY?

Ahmad Al Turk, MD* - Jieqi Liu, MD* - Umar Sharif Khawaja, MD - Sejal Kothadia, MD - David Vesole, MD * Equal Contributers

Presenter: Ahmad Al Turk MD, Tulane School of Medicine

Background: The Bone Marrow Transplant Survivor Study indicates that two-thirds of hematopoietic stem cell transplantation (HSCT) survivors will develop a chronic health condition. Arrhythmia has been identified as a complication of HSCT in several single-institution reports, but conclusive data at the national level is lacking.

Objective: Our study aims to identify the true incidence of arrhythmias, predisposing comorbidities and associated inpatient outcomes in those who underwent HSCT in the United States between 2008-2012.

Methods: We queried the 2008-2012 Nationwide Inpatient Sample database and identified all hospital admissions where HSCT was performed, excluding those

Results: Out of a total sample size of 72,151 patients undergoing HSCT, there were 8,383 (11%) reported cases of arrhythmias. Autologous HSCT made up 59.7% of the total, but incidence of arrhythmias was comparable between autologous (11.7%) and allogeneic (11.24%) transplant. Development of arrhythmia was associated with higher average LOS (27.65 vs 23.59 days, p

Conclusion: More than 10% of all HSCT recipients in the U.S. develop arrhythmias during their hospital stay, which is associated with an increased mortality rate, LOS, and admission cost. A high incidence of arrhythmias is seen in those with prior cardiovascular disease, with the highest noted in those with CHF. Further evaluation is needed to risk stratify cardiac patients prior to HSCT and cardiac monitoring may be warranted in high risk patients undergoing HSCT.

Poster #3b

COMPLEX CORONARY INTERVENTION IN A PATIENT WITH VON WILLEBRAND DISEASE; NOVEL USE OF VWF/FVIII CONCENTRATES SUPPLEMENTATION PRIOR TO PROCEDURE

Ahmad Al Turk, MD - Asaad Nakhle, MD - Mehul Bhalja, MD - Ahmad Jabbar, MD - Gerard Chaaya, MD - Cindy Leissinger, MD - Charisse Ward MD MPH
Presenter: Ahmad Al Turk MD, Tulane School of Medicine

Introduction: Optimal management of coronary artery disease (CAD) in patients with von Willebrand disease (VWD) is a rare but challenging clinical scenario given the risk of potential bleeding and the lack of evidence-based guidance. We present a case of a patient with VWD who underwent successful complex percutaneous coronary intervention (PCI).

Case Presentation: 65-year-old man with history of VWD Type 2B and CAD post remote 4-vessel coronary artery bypass grafts (CABG) underwent elective coronary angiography for new onset ventricular tachycardia. Laboratory work-up showed a platelet count of 51,000 per micro-liter and a PFA closure time of 246 seconds. Von Willebrand factor (VWF) antigen was 40%, VWF activity was 25%, and factor VIII (FVIII) activity was 61.9%. Patient underwent successful PCI of venous graft to right coronary artery then staged PCI of left main and ostial circumflex lesions. One hour prior to the procedures, he received one unit of leukoreduced platelets and 35 units per kilogram of VWF (using a VWF/FVIII concentrate) which was calculated to raise his functional VWF level to ~ 100%. Patient received aspirin, clopidogrel, and procedural heparin without any bleeding noted.

Discussion: When presenting with acute coronary syndrome, patients with VWD are more likely to receive medical therapy without PCI compared to patients who do not have a bleeding disorder. Limited cases where PCI was performed in patients with VWD have been reported. Supplementation with VWF/FVIII prior to the procedure has been described in a few patients undergoing CABG. However, to our knowledge, this is the first report on supplementation of VWF/FVIII and platelets prior to PCI in a patient with Type 2B VWD. This combination appears to be safe and effective in patients requiring elective complex coronary interventions. It should be noted that the half-life of VWF is approximately 12-18 hours, and that the patient will return to their baseline deficiency state approximately 24 to 36 hours after a single dose of VWF/FVIII, so continuation of anticoagulants or anti-thrombotics will increase the risk of bleeding as the VWF levels fall.

Conclusion: Complex coronary interventions can be safely performed in patients with VWD after VWF replacement using VWF/FVIII concentrates and platelet transfusion 1 hour prior to procedure.

Poster #3c

A SUCCESSFUL CONGESTIVE HEART FAILURE MANAGEMENT PROGRAM: QUALITY IMPROVEMENT INITIATIVE BY INTERNAL MEDICINE RESIDENTS

Naga Sai Shravan Turaga MD, Shashitha Gavini DO, Himani Manthena MD, Samiya Yasin MD, Jay Patel MD, Gregory Jacob MD, Imran Shuja MD, Ashwin Narayana MD, Syed Saad MD, Avaneesh Jakkoju MD, Elizabeth Borrero MD

Presenter: Naga Sai Shravan Turaga MD, LSU Health New Orleans

Background: Congestive heart failure (CHF) is a major healthcare issue associated with a high 30-day readmission rate. However, description of the causes and preventability of readmissions are lacking.

Objective: We performed a study to understand the factors causing CHF readmissions and to formulate effective strategies to reduce readmissions.

Methods: A multidisciplinary team consisting of residents, staff, case managers, pharmacists, and nurses was formed, and the study was done in 4 phases. In the 1st phase, retrospective chart review of patients readmitted for CHF exacerbation within 30 days of discharge between July 2016 and December 2016 was done, and the potential causes of readmissions were analyzed. In the 2nd phase, interventions to address the high impact factors were formulated, and the most feasible ones were selected. Third phase was the implementation phase. Fourth phase involved analyzing the logistics and problems associated with project implementation as well as studying the impact of the interventions. Plan-Do-Check-Act methodology was used in each phase to help identify factors and processes that required change. Chi-square test was used to test the statistical significance of the study.

Results: Retrospective chart review in 1st phase revealed 1month readmission rate of 22 % and 1week readmission rate of 11%. Major patient related factors impacting readmissions were medication unaffordability (75.5%), noncompliance (28.5%), and transport (10%). Major system related factors were lack of 1week post discharge follow up (100%), medication reconciliation and patient education. Selected interventions were peer-to-peer education of the multidisciplinary team about their role, interventions, CHF management guidelines, home health evaluation and medication reconciliation, establishing 1-week post discharge clinic follow up, post discharge nurse phone call in 3 days, and revision of educational material. The interventions were implemented from August 2017. Six months after the interventions were implemented, 1month readmission was 8.1% ($p=0.04$) and 1week readmission rate was 1.3% ($p=0.02$).

Conclusion: Every medical facility has its own unique patient population and hurdles in providing health care. An individualized approach to identify and address those hurdles can direct towards successful strategies to reduce CHF readmissions.

Poster #3d

USE OF DIRECT-ACTING ORAL ANTICOAGULANTS FOR LEFT VENTRICULAR THROMBUS

Stephanie Elagizi-Youssef, Laura A. Fuller, Kristina Dupre
Presenter: Stephanie Elagizi PharmD, Ochsner Health System

Background: The development of left ventricular (LV) thrombus is one of the most concerning complications after myocardial infarction (MI) and in reduced ejection fraction heart failure (HrEF). Anticoagulation with warfarin has become the mainstay of treatment. At present there is a paucity of data to support the use of direct-acting oral anticoagulants (DOACs) for the treatment of LV thrombus.

Objective: The purpose of this study is to compare DOACs to warfarin for the treatment of LV thrombus.

Methods: This retrospective cohort, pilot study included patients ≥18 years with ECHO confirmed LV thrombus due to MI or HrEF at Ochsner Health System. Patients were stratified according to their treatment of LV thrombus with warfarin or DOAC therapy. The primary outcome of the study was resolution of LV thrombus at repeat ECHO imaging within 12 months from initiation of anticoagulation. Secondary outcomes included time to repeat ECHO, ejection fraction improvement from baseline to repeat ECHO, major bleeding and thromboembolic events.

Results: A total of sixteen patients met inclusion criteria; 12 patients in the warfarin group and 4 patients in the DOAC group. There was no statistically significant difference in resolution of LV thrombus on repeat ECHO imaging between the DOAC and warfarin groups at 12 months (75% vs 100%, respectively; p=0.25). No major bleeding or thromboembolic events were reported in either group.

Conclusion: This pilot study indicates that DOACs may be a practical option for treatment of LV thrombus, and gives merit to the importance of continuing to evaluate the efficacy and safety of DOACs for this indication, though larger studies are needed to assess the effects of these agents on clinical outcomes.

Poster #4a

AN UNUSUAL ETIOLOGY OF CHEST PAIN IN A YOUNG PATIENT

Naga Sai Shravan Turaga M.D., Dedric Clarke M.D., Neeraj Jain M.D.
Presenter: Naga Sai Shravan Turaga MD, LSU Health New Orleans

Introduction: Myocardial ischemia is an uncommon cause of chest pain in those entering the third decade of life. More common etiologies include myocarditis, toxins, and hypertrophic cardiomyopathy. We present here a less common etiology of chest pain in a young patient.

Case Presentation: A 20-year-old male with a history of recent viral meningitis presented with complaints of chest pain after playing soccer. He had an episode of substernal heaviness radiating to left arm, palpitations and dyspnea lasting for a few minutes. He reported having similar episodes in the past which resolved with treatment in the emergency room. Physical exam was significant for marked resting tachycardia with heart rate of 220 beats/min. He was diagnosed with supraventricular tachycardia which resolved following treatment with 2 doses of adenosine and a dose of diltiazem. EKG after treatment showed sinus bradycardia, left ventricular hypertrophy and ST depressions in 8 of 12 leads. Initial troponin was 0.04 rising to 4.57. Coronary angiography showed a mid left anterior descending artery myocardial bridge. Echocardiogram did not show any significant structural or functional abnormalities. Electrophysiology study showed orthodromic atrio-ventricular reentrant tachycardia (AVNRT) with a concealed left lateral accessory pathway. Radiofrequency ablation was performed, and he was discharged on metoprolol succinate 25 mg daily. His symptoms were resolved at 1-month clinic follow up.

Discussion: Myocardial bridging (MB) is an anatomical anomaly in which segments of coronary arteries travel below the epicardial surface within the myocardium and thus subject to myocardial squeezing potentially limiting forward flow during systole. Such intramyocardial vessels can result in anginal symptoms especially during tachycardia or high contractile duration states. The reported incidence by invasive coronary angiography is up to 16.0% and even higher by CT angiography (~25%). MB is congenital and usually asymptomatic, but hemodynamics may be affected depending on the depth, length and orientation of the intramyocardial segment in relation to myocardial fibers. We believe myocardial bridging in the setting of marked SVT lead to acute myocardial infarction in our patient. Management options include beta blockers, calcium channel blockers, coronary stents, coronary bypass grafting, and surgical myotomy.

Conclusion: Less commonly recognized clinically, intramyocardial bridging should be considered as an etiology for ischemic chest pain in the appropriate clinical setting (eg, tachyarrhythmia) when the workup for traditional causes is otherwise negative.

Poster #4b

Spontaneous Pneumothorax with Pneumomediastinum and Pneumopericardium after dual chamber pacemaker placement

Jonathan Massey MD, Lina Ya'qoub MD, Khaldia Khaled MD
Presenter: Jonathan Massey MD, LSU Health Shreveport

Introduction: Pneumothorax, pneumopericardium and pneumomediastinum are disorders characterized by the presence of free air or gas in the related spaces.1 The combination of the above three presenting together is a very rare condition. We present a unique case with all three of the radiological findings in a 77-year-old male.

Case Presentation: A 77-year-old black male with a history of hypertension, type 2 diabetes mellitus and hyperlipidemia presented to our facility for permanent pacemaker placement (PPM) due to sick sinus syndrome. The patient underwent the pacemaker placement under standard procedures with insertion into the subcutaneous pocket in the left subclavian area. The follow up chest x-ray directly after pacemaker placement showed stable findings. Roughly five hours later, the patient developed severe left sided peri-orbital edema and swelling at the incision site of the pacemaker placement, followed by complaints of pleuritic chest pain with notable subcutaneous crepitus. The patient's vitals remained stable. A chest x-ray showed the pacemaker in the left dorsal temporal region with two leads in the expected location of the right atrium and ventricle. There was however a 10-15% left apical pneumothorax with extensive subcutaneous emphysema as well as a noted pneumopericardium with pneumomediastinum. There was no evidence of the pacer leads piercing the myocardium or extension into the pericardial space. The patient required close observation in the MICU for three days. Daily chest x-rays showed stable improvement of the pneumothorax, pneumomediastinum and pneumopericardium. The patient was discharged in stable condition and seen for outpatient follow up with repeat chest x- ray showing complete resolution of the above mentioned.

Discussion: Approximately 180,000 patients undergo PPM implantation in the United States each year. 2 Pacemaker implantations is usually considered a safe procedure, though acute complications do occur in 4%-7% of cases. Most frequently consisting of lead displacement, traumatic pneumothorax, hemopneumothorax and pericardial tamponade. 3 We present this case because of our patient's unusual clinical presentation. Our literature search did not reveal a patient presentation where an association between a pneumothorax, a pneumomediastinum and pneumopericardium were all present at the same time. While there have been cases where two out of the above three may be present, frequently those patients had underlying anatomical defects, of which our patient had none. Our case also presents an elusive etiology in that there was no diagnostic evidence of lead migration or perforation. Lastly, our case highlights that conservative non-surgical treatment may be sufficient in patients with stable vital signs and stable lead parameters.

Conclusion: While PPM is a commonly performed procedure, this case demonstrates that unusual and rare complications do exist.

Poster #4c

A RARE CASE OF ADULT CONGENITAL HEART DISEASE: SINGLE VENTRICULAR CHAMBER WITH ANOMALOUS RIGHT CORONARY ARTERY

Muhammad Shabbir Rawala, MD, Syed Bilal Rizvi, MD
Presenter: Muhammad Shabbir Rawala MD, Rapides Regional Medical Center

Introduction: Patients with single-ventricle physiology encompass a wide array of anatomic subtypes, including but not limited to: tricuspid atresia, pulmonary atresia, hypoplastic left heart syndrome, double-outlet or double-inlet ventricles. The outcomes for patients with single ventricle born before 1990 are relatively poor. We present a unique case of an 81-year-old who had survived with single ventricular chamber physiology without any corrective surgery.

Case Presentation: An 81-year-old female with history of arthritis, diabetes mellitus type II, coronary artery disease, hypertension presented to the hospital as non-ST elevation myocardial infarction (NSTEMI). She was started on antiplatelet and anticoagulation. Echocardiogram revealed ejection fraction to be 40-45% and one single ventricle which was thought to be left ventricle with possible transposition of great vessels.
The patient then underwent left heart catheterization that identified that patient has single ventricle and anomalous origin of the right coronary artery. She was also found to have double vessel coronary artery disease with diffuse stenosis of mid-RCA at 80% and proximal circumflex at 95%. She was managed conservatively as was high risk for CABG given her rare congenital condition.

Discussion: Patients with single ventricle are at risk of long-term morbidity, including heart failure, neurological injury, multisystem organ failure, and early death. The mortality risk of these patients is high as most of the patients without corrective surgery do not proceed to adulthood. Those who proceed to adulthood mostly develop NYHA functional class III heart failure symptoms. Many of the patients are functional; however, they do suffer from limited exercise tolerance given their persistent cyanosis. Our case did have secondary polycythemia due to chronic hypoxia developed as a result of patient's cyanotic congenital heart condition. She was perfectly well with good mentation with oxygen saturation of 80-88%.

Our case had multivessel coronary artery stenosis along with a rare presentation of congenital heart disease in adulthood. The patient was at high risk for coronary artery bypass due to her cardiac morphology. The patient was offered percutaneous coronary intervention, but she declined and chose to be treated conservatively with only medical management.

Conclusion: We present a rare case of an elderly female surviving with a single ventricular chamber. The patient is an exception to the usual process of the pathology as most patients without corrective surgery seldom survive into adulthood

Poster #4d

ATRIAL MYXOMA MIMICKING MITRAL VALVE STENOSIS LEADING TO PULMONARY HYPERTENSION

Muhammad Shabbir Rawala, MD, Syed Bilal Rizvi, MD
Presenter: Muhammad Shabbir Rawala MD, Rapides Regional Medical Center

Introduction: Cardiac myxoma is the most common type of primary cardiac neoplasm and accounts for 30%–50% of all primary tumors of the heart with an annual incidence of 0.5 per million population. Over 70% of all cardiac myxomas originate from the left atrium and 18% from the right atrium. The clinical presentation varies among patients depending on the size, mobility, and location of the tumor. Most myxomas present with one or more effects of a triad of constitutional, embolic, and obstructive manifestations. Systemic embolism can occur in 25-50% of left atrial myxomas.

Case Presentation: A 48-year-old female with past medical history of diabetes mellitus, hypertension, and hypothyroidism presented to the cardiologist's office with complaints of chest tightness, decreased exercise intolerance and dyspnea. She was initially treated as bronchitis by her primary care physician. The symptoms did not resolve; therefore, she was referred to a cardiologist for evaluation. The echocardiogram revealed a mass in the left atrium. Invasive angiography was performed to evaluate coronary artery disease; however, the angiography was negative for any significant stenosis. Cardiothoracic surgery was consulted for resection of myxoma. Patient's symptoms completely resolved after resection. Intraoperatively her pulmonary artery pressures decreased from 60/45 mm Hg to 25/10 mm Hg after resection.

Discussion: King first described left atrial myxoma in 1845. It was initially diagnosed by echocardiography in 1952 and resected for the first time in 1955. Sporadic cases present in the fifth decade while familial cases present in the second decade.
Cardiac myxomas present with systemic manifestations in 90% of the patients, characterized by weight loss, fatigue, fever, anemia (often hemolytic), and elevated sedimentation rate. Large left atrial myxoma in only 10% of patients can cause a mechanical obstruction causing functional mitral valve stenosis leading to left atrial dilatation and pulmonary hypertension. This phenomenon causes congestive heart failure like symptoms which improve after resection of myxoma as experienced in our case. Neurologic events are common in patients with myxoma and can occur in approximately one-third of these patients.

Imaging modalities such as echocardiography, cardiac computerized tomography or cardiac magnetic resonance imaging aid in confirming the clinical diagnosis. Surgical resection is recommended in order to prevent systemic embolic events and improvement of cardiac symptoms. The survival after total resection of myxoma is high. There is a 1% - 3% recurrence; therefore, long-term follow-up with echocardiography is recommended.

Conclusion: This case highlights the importance of being suspicious even in a young patient regarding the presence of myxoma which was causing her to have congestive heart failure symptoms and complete resolution after resection.

Poster #5c

ACUTE ST-ELEVATION MYOCARDIAL INFARCTION SECONDARY TO CORONARY VASOSPASM IN AN ADOLESCENT USING ANABOLIC-ANDROGENIC STEROIDS

Abdulaziz Joury
Presenter: Abdulaziz Joury MD, Ochsner Health System

Introduction: The use of anabolic-androgenic steroids (AAS) is increasing worldwide and its long-term use associated with myocardial dysfunction, significant atherosclerosis and cardiovascular events. AAS accelerate coronary atherosclerosis or by coronary vasospasm and predispose to acute myocardial infarction. Vasospastic etiology effect of AAS on coronary artery is most likely due to altering vascular nitric oxide system.

Case Presentation: A 19-year-old male who gained 15 pounds of muscle mass over two years of using androstenedione as AAS. He developed sudden onset of typical angina chest pain that lasted for more than 30 minutes before his arrival at the hospital. Initial electrocardiogram (ECG) showed ST-elevation myocardial infarction (STEMI) to 0.3 mV in V2 and V3. Emergent cardiac catheterization was done, and his angiogram showed right dominant coronary artery and showed normal coronary arteries. Initial troponin I of 1.3 ng/mL and peaked at ten ng/mL in the next set 6 hours. Transthoracic echocardiogram showed normal studies except a mild decrease in RV function. This case showed a presentation of vasomotor angina was made and most likely triggered by AAS.

Discussion: Coronary artery disease and hypercoagulable states might lead to imbalance between oxygen demand and supply and endothelial dysfunction. Strong coronary stimulants such as cocaine, cigarettes have been reported to increase the risk of coronary artery disease. One of the uncommon risk factors for vasospasm effect of the coronary arteries is AAS, and this is attributed to multiple factors. One of the most studies mechanisms is with decreased concentrations of arterial endothelial cyclic guanosine monophosphate which itself directly inhibited by AAS.

Conclusion: AAS is known to increase the risk of adverse cardiovascular events, AAS users education about cardiac risk is imperative.

Poster #5b

A CASE OF TRANSIENT CORTICAL BLINDNESS FOLLOWING CARDIAC CATHETERIZATION

Sicinschi S, Dahal K, Gonzalez-Toledo E, Njoku AU, Mina G, Modi K
Presenter: Stefan Sicinschi MD, LSU Health Shreveport

Introduction: Transient cortical blindness is a rare complication that can occur following administration of contrast media. It is characterized by partial or complete loss of perceived vision following administration of contrast. The symptoms are self-limited and usually resolve spontaneously within a few days.

Case Presentation: An 83-year-old male with coronary artery disease, history of coronary artery bypass surgery, hypertension, and diabetes presented with NSTEMI. He underwent coronary angiography with angioplasty and stenting of left circumflex and distal left main arteries. During the procedure he received 200 ml of Iodixanol (Visipaque), an iodine-containing non-ionic radiocontrast agent. After returning to the medicine floor he developed acute bilateral vision deficits with blurry vision and bilateral loss of visual acuity. He denied any associated flashes of light, floaters, color deficits, foreign body sensation, itching, pain, or photophobia. He reported the blurriness to be equal in both eyes. On physical examination his pupils were equal, reactive to light, without ocular discharge, erythema, or visual field deficits. The rest of his neurologic examination was normal. Dilated funduscopic examination performed by on-call ophthalmologist was negative for ocular ischemia. The patient's ocular discs, vessels, maculas, and peripheries were within normal limits. Non-contrast head CT imaging was remarkable for bilateral occipital hyperdensities with involvement of right thalamus (Figure 1). Initially there was concern for bilateral occipital hemorrhages. The patient was transferred to the intensive care unit for close monitoring of neurologic status. The following morning the patient's vision deficits had completely resolved. Repeat CT imaging showed resolution of the hyperdensities (Figure 1). The patient's symptoms and neuroradiologic changes were believed to be consequences of contrast administration. An MRI of the brain showed punctate ischemic lesions in the territory of the left posterior cerebral artery, however these were not thought to be responsible for the patient's symptoms as unilateral PCA stroke normally presents with homonymous hemianopia. The patient was eventually discharged home without further complications.

Discussion: This patient developed transient bilateral loss of visual acuity following percutaneous coronary intervention. His symptoms corresponded with bilateral occipital lobe hyperdensities visualized on non-contrast head CT. His symptoms and neuroimaging abnormalities spontaneously resolved in less than 24 hours. The presentation is consistent with contrast-induced transient cortical blindness, a rare complication that can occur after administration of ionic or nonionic contrast media (1,4). It is characterized by partial or complete loss of perceived vision following administration of contrast. On examination there are normal fundi, normal papillary reflexes, and unaltered extraocular movements (2,3). The reaction does not seem to be volume dependent (2), and it can occur after administration of both high and low-osmolarity iodinated contrast compounds, including Iodixanol which is what our patient received (11,12). The onset can occur from minutes to

hours after contrast administration, and the outcome is generally favorable, with complete recovery described as ranging from 12 hours to 5 days (3). The mechanism of injury has been postulated to be a form of posterior reversible encephalopathy syndrome (PRES) (5). It is postulated that due to decreased autoregulatory capacity, the blood-brain barrier is more permeable in the occipital cortex in the setting of uncontrolled hypertension, predisposing it for contrast extravasation and neurotoxicity (5,10). On non-contrast head CT, there is often enhancement from peri-procedural contrast administration which can mimic the appearance of intracerebral or subarachnoid hemorrhage (6,7). Although rare, recurrence of contrast-induced encephalopathy has been previously documented (11). Angiography involving internal mammary artery grafts carries a higher risk for transient cortical blindness due to the anatomical proximity to the vertebral arteries and involvement of posterior circulation (9). Transient cortical blindness is the most common manifestation of contrast-induced encephalopathy following cardiac catheterization, with 27 cases previously documented in literature; 13 of these cases occurred after diagnostic angiography involving internal mammary grafts, and 7 occurred after PCI (9).
Due to concern for hemorrhage, the reaction may lead to discontinuation of antiplatelet or anticoagulation therapy. In the setting of coronary angiography this can have detrimental effects. If there is clinical suspicion for contrast-induced reaction, MRI can be used to guide therapy as it can distinguish blood and ischemia from contrast medium. Our patient had an MRI which did not show bleeding or ischemic lesions consistent with the patient's neurologic deficits. It showed lacunar infarcts in the territory of the left posterior cerebral artery. The infarcts were likely a consequence of the cardiac catheterization, but they were unlikely to have caused the patient's symmetric loss of visual acuity and blurriness; unilateral ischemia in territory of posterior cerebral artery normally presents as homonymous hemianopia. It is possible that under different circumstances the lacunar infarcts would have gone unnoticed as the incidence of asymptomatic radiographic infarcts following cardiaccatheterization has been shown to be as high as 8% (8). Incidental infarcts have also been previously documented in other cases
involving transientcortical blindness (1,11).

Conclusion: Transient cortical blindness is a rare complication following administration of ionic or nonionic contrast during coronary angiography. The associated lesions on non-contrast CT may mimic the appearance of subarachnoid or intracerebral hemorrhage. The symptoms and imaging abnormalities usually resolve spontaneously within a few days. MRI can be used to guide therapy as it can distinguish blood and ischemia from contrast medium.

Poster #5c

STRESS CARDIOMYOPATHY FOLLOWING INTRAVENOUS EPINEPHRINE ADMINISTRATION

Tasleem Katchi, Jasjit Bhinder, Anthon Fuisz, Wilbert S. Aronow, Diwakar Jain
Presenter: Tasleem Katchi MD, Tulane School of Medicine

Introduction: Stress cardiomyopathy, also referred to as Takotsubo cardiomyopathy, is characterized by acute systolic dysfunction, which is typically reversible. The condition is usually precipitated by extreme emotional or physical stress, however, may occur even in the absence of the same. Intravenous (IV) epinephrine is frequently used in clinical practice during the resuscitation of patients with cardiac arrest, cardiogenic shock, hypotension and anaphylaxis. Few cases of stress cardiomyopathy following the use of IV epinephrine have been reported. Presentation may mimic acute coronary syndrome (ACS). This may pose a diagnostic and therapeutic dilemma, especially in critically ill patients.

Case Presentation: An 80-year old female was admitted to the intensive care unit for acute bowel perforation and pneumoperitoneum. She underwent exploratory laparotomy with small bowel resection and surgical anastomosis. Her post-operative course was complicated by hypoxic respiratory failure requiring mechanical ventilation. While on the ventilator, she had an episode of severe respiratory distress, unresponsiveness and hypotension. As part of her resuscitation, she was administered 1 mg of IV epinephrine. The acute episode resolved with the administration of IV epinephrine, vecuronium and fluids. Cardiac biomarkers were obtained due to concern for ACS. Troponin I level was 0.06 ng/ml with a peak of 2.16 ng/ml. An electrocardiogram (ECG) revealed no acute ischemic changes. A transthoracic echocardiogram (TTE) revealed moderately reduced systolic function with akinetic apical and hyperkinetic basal segments. This was unlike the prior TTE which showed normal systolic function and no wall motion abnormalities. Clinical picture was concerning for ACS. Due to the patient's critical condition and inability to anticoagulate, she was conservatively managed with low-dose aspirin and beta-blocker. Repeat TTE a week later showed complete recovery of systolic function and resolution of wall motion abnormalities.

Discussion: The most commonly accepted hypothesis for the pathogenesis of stress cardiomyopathy is catecholamine-induced microvascular dysfunction with resultant myocardial stunning. This is supported by the presence of excess catecholamine levels in patients with stress cardiomyopathy compared to patients with A1C1S1.1 1E1n1d1o1m1y1o1c1a1r1d1i1a1l1 1b1i1o1p1s1i1e1s1 1o1f1 1p1a1t1i1e1n1t1s1 1w1i1t1h1 1s1t1r1e1s1s1 1c1a1r1d1i1o1m1y1o1p1a1t1h1y1 1h1a1v1e1 1s1h1o1w1n1 1a1 1p1a1t1t1e1r1n1 1o1f1 1m1o1n1o1n1u1c1l1e1a1r1 1i1n1f1i1l1t1r1a1t1i1o1n1 1a1n1d1 1c1o1n1t1r1a1c1t1i1o1n1 1b1a1n1d1 1n1e1c1r1o1s1i1s1,1 1s1e1e1n1 1i1n1 1c1l1i1n1i1c1a1l1 1s1t1a1t1e1s1 1o1f1 1c1a1t1e1c1h1o1l1a1m1i1n1e1 1e1x1c1e1s1s1.1 1F1u1r1t1h1e1r1m1o1r1e1,1 1s1t1r1e1s1s1 1c1a1r1d1i1o1m1y1o1p1a1t1h1y1 1t1y1p1i1c1a1l1l1y1 1p1r1e1s1e1n1t1s1 1w1i1t1h1 1h1y1p1o1k1i1n1e1s1i1s1/1a1k1i1n1e1s1i1s1 1o1f1 1t1h1e1 1a1p1i1c1a1l1 1v1e1n1t1r1i1c1u1l1a1r1 1s1e1g1m1e1n1t1s1 1a1n1d1 1h1y1p1e1r1k1i1n1e1s1i1s1 1o1f1 1t1h1e1 1b1a1s1a1l1 1s1e1g1m1e1n1t1s1,1 1r1e1s1u1l1t1i1n1g1 1i1n1

2a2p2i2c2a2l2 2b2a2l2l2o2o2n2i2n2g2.2 2T2h2i2s2 2c2a2n2 2b2e2 2a2t2t2r2i2b2u2t2e2d2 2t2o2 2t2h2e2 2v2a2r2i2a2b2l2e2 2d2i2s2t2r2i2b2u2t2i2o2n2 2o2f2 2c2a2t2e2c2h2o2l2a2m2i2n2e2 2r2e2c2e2p2t2o2r2s2 2i2n2 2t2h2e2 2v2e2n2t2r2i2c2l2e2.2 2T2h2u2s2,2 2e2x2o2g2e2n2o2u2s2 2e2p2i2n2e2p2h2r2i2n2e2 2c2a2n2 2p2r2e2c2i2p2i2t2a2t2e2 2s2t2r2e2s2s2 2c2a2r2d2i2o2m2y2o2p2a2t2h2y2 2w2i2t2h2 2t2h2e2 2s2a2m2e2 2m2e2c2

Conclusion: IV epinephrine can result in stress cardiomyopathy which resolves spontaneously over days. Increased awareness of this condition amongst physicians can help avoid invasive interventions, thereby minimizing cost and complications.

Poster #5d

SUCCESSFUL REPAIR OF A LARGE ABDOMINAL AORTIC ANEURYSM USING AN ENDOVASCULAR APPROACH

Tasleem Katchi, Ahmad Jabbar, Charisse Ward
Presenter: Tasleem Katchi MD, Tulane School of Medicine

Introduction: With the introduction of the newer generation endovascular (EV) grafts, the anatomic limitations for the use of an EV approach for abdominal aortic aneurysm (AAA) repair have been minimized. About 80% of AAA in the United States are repaired with an EV approach. We present a case of the successful repair of a large AAA using an EV approach.

Case Presentation: A 56-year old male with a history of coronary artery disease, hypertension, and remote tobacco use presented with a pulsatile sensation in the abdomen. Computed Tomography angiogram of the abdomen/pelvis demonstrated a fusiform AAA measuring 10.7 cm (anteroposterior) x 11.8 cm (transverse) x 13.5 cm (longitudinal) extending to the aortic bifurcation with extensive mural thrombus and calcifications. The common iliac arteries were ectatic with multifocal calcified plaque. After extensive discussion of the risks and benefits of surgical versus EV approach, the decision was made to proceed with an EV approach and the AAA was repaired using the GORE EXCLUDER DEVICE (main body 35 x 14.5 x 140 mm) with bilateral common iliac artery extensions. The patient tolerated the procedure well without any immediate complications.

Discussion: The choice of an EV versus open approach for AAA repair is influenced by aneurysm factors such as the size and anatomy of the lesion, and patient factors such as age, comorbidities and the surgical risk. Although short term morbidity and mortality have been shown to be lower with the EV approach, significant difference in the long term outcomes has not been demonstrated. EV repair carries the risk of future aneurysm rupture and surveillance of the EV stent involves radiation exposure. The EV approach is favored in patients with limited life-expectancy and high surgical risk. However, the use of this approach in young patients with low to intermediate surgical risk and favorable anatomy remains controversial.

Conclusion: The decision for an EV versus open approach for AAA repair should involve evaluation of individual factors and a comprehensive discussion of the same with the patient.

Poster #6a

A RARE CASE OF GIANT LEFT ATRIUM

Asaad Nakhle, MD; Ahmad Al-Turk, MD; Ahmad Jabbar, MD; Charisse Ward, MD
Presenter: Nakhle MD, Tulane School of Medicine

Introduction: Giant left atria (GLA) was first described in 1849 by Hewett, but is rare in the era of modern medicine. We present a case of symptomatic GLA.

Case Presentation: The patient is a 70-year-old female with a long history of rheumatic mitral valve disease who underwent mitral valve replacement with a St. Jude's mechanical valve. She presented with progressive shortness of breath, diminished exercise tolerance, palpitations, difficulty swallowing, increased abdominal girth, and cachexia.

She underwent a chest x-ray which showed significant cardiomegaly with the right heart border reaching the right chest wall. Her chest radiograph and CT scan revealed a markedly enlarged cardiac silhouette with a massively dilated left atrium and chronic partial atelectasis of the right middle and lower lung lobes. She underwent a two-dimensional transthoracic echocardiogram (TTE) which revealed a left atrial diameter of 10.9 cm and an indexed left atrial volume of 1200 ml/m2.

Discussion: The patient's long-standing mitral valve disease resulted in chronically elevated left atrial pressure. This led to an increase in compliance leading to giant left atrium which compressed the lung and esophagus causing shortness of breath and dysphagia; respectively. Surgical correction was explored, but she was found to be a poor surgical candidate due to multiple comorbidities.

Conclusion: GLA is characterized by a left atrial anteroposterior diameter greater than 8 cm on TTE. It is most commonly found in patient with rheumatic valvular disease, but it has also been described in patients with other forms of mitral valve disease. This includes chronic atrial fibrillation, left ventricular failure, patent ductus arteriosus, and ventricular septal defect.

Patients with GLA develop symptoms due to the enlarged atrium, development of heart failure, or compression of surrounding structures. They manifest as shortness of breath, paroxysmal nocturnal dyspnea, palpitations, atrial fibrillation, thromboembolic events, or dysphagia. Surgical management is indicated during the time of mitral valve surgery if the patient has evidence of intracardiac or extracardiac compressive symptoms or recurrent thromboembolic events.

Poster #6b

A RARE PRESENTATION OF SYNTHETIC MARIJUANA USE AND SUBSEQUENT DOUBLE INFARCTION OF THE RIGHT CORONARY ARTERY AND LEFT CORONARY ARTERY THROMBOTIC

Ladyzhenskiy E. MD, Subramanian P. MD
Presenter: Edward Ladyzhenskiy MD, LSU Health New Orleans

Introduction: Synthetic marijuana use has been growing exponentially but data and research regarding the health effects is scarce. Cardiac arrest is a poorly understood consequence of its use. Dual coronary artery thrombosis is a rare clinical and angiographic entity that typically leads to cardiogenic shock or even sudden cardiac death. Despite reports of multiple ruptured plaques with thrombus formation have been reported in greater than 10% of autopsied cases, concomitant thrombosis and occlusion of two coronary arteries continues to be uncommon finding on angiography.

Case Presentation:
We present a case of a 32-year-old African American male that presented as a cardiac arrest after smoking synthetic marijuana earlier that day. He arrested en route with initial rhythms of recurrent ventricular tachycardia and ventricular fibrillation necessitating multiple defibrillation attempts before return of spontaneous circulation (ROSC) was obtained. His only significant medical history is daily marijuana use but denied any other substance abuse. Post-resuscitation blood pressure was 151/68mmHg and heart rate of 104 beats per minute with a temperature of 98.9F. He was intubated on arrival with saturation of 99% on 60% FIO2. His post-resuscitation electrocardiography (EKG) revealed acute inferior and antero-lateral ST-elevation. A transvenous pacemaker was placed in the emergency room and the patient was taken for emergent angiography. Focus was taken on the suspected culprit right coronary artery (RCA). He was found to have an acute occlusive thrombus with contrast staining at the mid-RCA and thrombolysis in myocardial infarction (TIMI) flow 0 past the lesion. He was started on bivalirudin infusion and given ticagrelor 180mg with aspirin 325mg load through his oral-gastric tube (OGT). This was treated with balloon and placement of a single drug-eluting stent (DES). Attention was then drawn to the left anterior descending artery with finding of an acute thrombosis with contrast staining past the first septal artery and TIMI 0 flow past the lesion. The lesion was treated with balloon angioplasty and a single DES placement. The left circumflex artery was free of any disease. An intra-aortic balloon pump was placed for hemodynamic support post-intervention. He was subsequently transferred to the intensive care unit and initiated on hypothermic protocol. Initially laboratory studies revealed a pH of 6.9, pCO2 72.1, pO2 14, pHCO3 15.4, with a base excess of -14. His white blood cell count was 15.9K/uL, hemoglobin of 13.5g/dL, lactic acid 7. 4mmol/L, potassium of 5.4mmol/L, creatinine 1.4mg/dL, troponin of 0.24ng/mL, brain natriuretic peptide (BNP) 23pg/mL, and had a negative urine drug screen. Post-procedure chest x-ray revealed bilateral pulmonary edema, no pleural effusions with a normal cardiac silhouette. Transthoracic echocardiography demonstrated antero-septal dyskinesis with a depressed ejection fraction of 30% with no left ventricular thrombus identified. The following EKG noted resolution of his ST elevations but profound Q-waves in the inferior and antero-lateral leads and frequent ventricular ectopy. His troponin rose to over 50ug/dL (normal:

Discussion: Synthetic marijuana products have been on the market for many years and are increasingly popular among young Americans. They are poorly government regulated herbs that are often laced with various chemical analogues aimed to mimic the effects of tetrahydrocannabinol (THC). Many of these chemicals have a higher binding affinity to CB-1 and CB-2 receptors than THC and therefore can result intense systemic effects and profound cardiac side effects. These substances typically cannot be detected on routine toxicology screens and discovery is often made through patient acknowledgement. Some previously described side effects range from severe psychotic episodes and hallucinations to seizures and heart attacks. The link between synthetic marijuana use and heart attacks has been rarely observed. Our presentation reinforces the dangers of synthetic marijuana use and the cardiac risks associated with its use. In addition, acute myocardial infarction of two vascular territories or a "double infarction" is very rare phenomenon. In a series of 711 patients by Pollak et al. who underwent primary PCI, 18 or 2.5% of cases were multiple culprit arteries. The exact cause for this event is unknown but various explanations have been proposed. They include marked systemic inflammatory and catecholamine surge; hemodynamic instability with hypotension leading to blood stasis and acute occlusion of different vessels; prolonged vasospasm; coronary embolization or arteritis; and hypercoagulable conditions leading to thrombosis. In retrospective analysis studies of non-synthetic marijuana consumption, the risk of acute myocardial infarction increases 3-8% irrespective of consumption duration. Studies attribute this to toxic effects of THC generating more carboxyhemoglobin that could interfere with cellular oxygenation and ultimately the increased cardiovascular risks such as myocardial infarction. Cocaine induced myocardial infarctions are predominantly related to vasospasm with or without exacerbation of underlying coronary artery disease. It is very difficult to prove the exact etiology as it pertains to our particular case but hypotension and blood stasis appears to be the most probable etiology. The preferred method of management, whether thrombolysis, primary PCI, or both, remains undetermined. Given the acuity of the presenting circumstance, primary PCI would appear to be the preferred and definite method of treatment. Despite emergent treatment, about one third of patients will still expire, supporting the need for aggressive management and upfront care.

Conclusion:

Synthetic marijuana is an ever-growing and popular consumer product available to most without much research and regulation. The list of dangerous effects is on rise and is more evident with greater recreational use. The association between these synthetic products and cardiovascular complications such as arrhythmias, myocardial infarction, and death continue to raise questions of their consumer benefit, if any. Little is understood between their direct effects on myocardial infarction particularly in the young population. Even less is understood about dual vessel myocardial infarctions and how they relate to these toxic substances. This case demonstrates two very rare entities in cardiovascular medicine and should raise more awareness in the clinical correlation of both.

Poster #6c

ANOMALOUS LEFT CORONARY ARTERY ARISING FROM THE NON-CORONARY CUSP

Carolina Larmeu MD, Khush Parikh MD, Lauren Haddad MD
Presenter: Carolina Larmeu MD, Our Lady of the Lake Children's Hospital, Pediatric Residency Program

Introduction: Congenital anomalies of the coronary arteries are defined as variations in coronary anatomy that occur in

Case Presentation: This is a case report of a 15 year old female athlete who presented with chest pain atypical of cardiac etiology and an incidence of syncope while running in a track meet. Original echocardiogram showed a normal heart with normal contractility although the coronaries were not described. A treadmill stress test elicited nonspecific ST changes with J-point depression in early recovery. Anomalous take-off of the LCA from the non-coronary cusp of Valsalva was subsequently demonstrated on cardiac CT. The patient was then sent to Boston Children's Hospital where she underwent marsupialization of the anomalous LCA which was found to have an intramural course. Echocardiogram at 3 months post-op noted to be normal.

Discussion: In this patient, origin of the LCA from the noncoronary sinus of Valsalva was shown to predispose to symptoms suggestive of ischemia.

Conclusion: Although rare, several case reports of patients suffering severe consequences from LCA from the noncoronary sinus of Valsalva show that this anomaly should not be deemed as benign. Although rare, the importance of recognition of the anomaly is in the ability of physicians to manage its condition. Further description of this anomaly is needed to allow for improved recognition and diagnosis, which may lead to prevention of SCD, especially in young athletes.

Poster #6d

CASE OF ELDERLY PATIENT WITH VENTRICULAR SEPTAL DEFECT

Mehnaz Rahman MD, Elias Hanna MD, D Luke Glancy MD
Presenter: Mehnaz Rahman MD, LSU Health New Orleans

Introduction:
A 66-year-old man presented with a moderate-sized ventricular septal defect and severe pulmonary hypertension that was responsive to vasodilator therapy. His ECG demonstrated
biatrial enlargement and biventricular hypertrophy. The elderly presentation is unusual for this type of shunt.

Case Presentation: A 66-year-old man presented to the hospital with biventricular failure manifested by
dyspnea, elevated jugular venous pressure, hypoxemia, peripheral edema, and pulmonary edema on his chest radiograph. A 4/6 holosystolic murmur was heard at the left sternal border and was accompanied by a systolic thrill. A faint diastolic murmur of Graham Steell could be heard. The electrocardiogram (ECG) was abnormal (Figure 1). Echocardiography showed a moderate size (1 cm in diameter with a 2.6 cm aortic ring diameter) perimembranous ventricular septal defect (VSD), biventricular hypertrophy with a preserved left ventricular ejection fraction, and marked left atrial enlargement. Doppler assessment revealed bidirectional flow across the VSD. Cardiac catheterization demonstrated severe pulmonary arterial hypertension with a pressure of 106/30 mmHg when the systemic arterial pressure was 134/78 mmHg. The pulmonary/systemic flow ratio (QP/QS) was 1.4. Pulmonary vascular resistance (PVR) was 9 Wood units. With the administration of inhaled nitric-oxide, pulmonary vascular resistance decreased to 3.75 Wood units. The shunt became exclusively left-to-right; QP/QS increased to 3; and systemic arterial oxygen saturation increased from 92% to 100%. His coronary arteriography was normal. The patient received a loop diuretic and dual vasodilator therapy. He improved and was discharged with an appointment to return for surgical closure of his VSD. Unfortunately, he did not return for his operation.

Discussion: Small VSDs frequently close spontaneously and often are hemodynamically insignificant when they persist. Large VSDs surgical intervention due to symptom development well before adulthood, frequently in infancy. This case is unique in that the patient presented with a hemodynamically significant VSD at the age of 66. Those years of increased pulmonary blood flow and pressure undoubtedly increase his operative risk for closure of the defect, but he has returned to the hospital with another episode of cardiac failure, suggesting that “conservative” management may have more risk than surgery.

Figure 1: Katz-Wachtel Pattern on EKG

-There is sinus tachycardia at a rate of 103 beats/minute.
-Broad and tall P waves in the inferior leads and biphasic P waves with large positive and negative components in lead V1 indicate biatrial enlargement.

-Left axis deviation of the QRS in the frontal plane with small q waves in leads I and aVL suggest left anterior fascicular block, but the R waves in those leads are smaller than is usually the case with left anterior fascicular block.
-R in lead aVR > 5 mm, when 5 mm = 0.5 mV indicates right ventricular hypertrophy.5 R aVL + SV3 > 22 mm(2.2 mV) indicates left ventricular hypertrophy
-The diagnosis of biventricular hypertrophy is supported by the large biphasic QRSs in the mid and lateral precordial leads. These were originally described in patients with ventricular septal defect, but may be seen in patients with other causes of biventricular enlargement
- The tracing meets criteria for right bundle branch block, and the fragmented rsR´S´ pattern in lead V1 suggests a higher pulmonary arterial pressure than when the more common rSR´ pattern is present.
-The widespread ST-T changes are part of the pattern of biventricular enlargement.

Conclusion: Medical and surgical innovations have allowed more children with congenital heart defects to survive into adulthood. Those presenting without having had any previous intervention are still rare.

It is important as general cardiologists to familiarize themselves with their hemodynamic consequences and unique diagnostic findings in order to facilitate timely therapeutic intervention.

Poster #7a

CHEMOTHERAPY INDUCED PERICARDITIS

Tejas Joshi MD, Anna Plemmons MS4, Ryley McPeters MD, Eugene Koffman MD, Frank Smart, MD, FACC
Presenter: Tejas Joshi MD, LSU Health New Orleans

Introduction: 5-fluoruracil (5-FU) is an anti-metabolite commonly used in chemotherapeutic regimens. FOLFOX, a combination of Folinic acid, Oxaliplatin, and 5-FU, has been implicated in only a few cases of cardiac toxicity ever reported. 5-FU, both alone and as a part of combination chemotherapy, has been associated as an extremely rare cause of acute pericarditis. We present a case of 5-FU induced pericarditis within 24 - 48 hours of FOLFOX induction.

Case Presentation: A 55-year-old woman with gastroesophageal adenocarcinoma diagnosed on low-dose screening chest CT presented to the ED with progressive, 8/10 substernal pressure-like pain. Initial workup ruled out PE, CHF exacerbation, and STEMI. Initial EKG revealed NSR, diffuse ST segment elevations in at least eight leads. Elevated J points, along with diffuse PR segment depressions were visualized, which subsequently resolved with supportive care after discontinuation of 5-FU. This patient was determined to have 5-FU induced pericarditis.

Discussion: Cardiac toxicity has an incidence of 1.6% of the patients treated with 5-FU, with 69% of events occurring during or within 72-hours of treatment.69% of studied populations reveal ischemia or ST-T wave changes, cardiac death occurs in less than 1%, and only 1.4% of events are found to be pericarditis.

Conclusion: Determining if EKG changes and chest pain are related to pericarditis or some other electrical abnormality in these cases can prove difficult. If cardiotoxicity occurs at anytime during 5-FU therapy it should be discontinued and a new alternative chemotherapy regimen should be started. Failure to recognize pericarditis in this patient population results in delayed treatment, and potentially poorer healthcare outcomes.

Poster #7b

DESPERATE TIMES REQUIRE DESPERATE MEASURES: A PATIENT PRESENTING WITH LEG PAIN

Solomon Seifu, MD; Zola N'dandu, MD
Presenter: Solomon Seifu MD, LSU Health New Orleans

Introduction: Critical limb ischemia is the end stage of peripheral arterial disease presenting with rest pain, severe ischemic ulceration or tissue gangrene of the extremities. The overall mean annual prevalence of PAD is 10.69% and CLI is 1.33% with CLI representing 11.08% of total PAD annually. Patients with critical limb ischemia have a high risk of limb loss, increased mortality rates and decreased quality of life. Treatment involves revascularization with surgical bypass or percutaneous transluminal angioplasty (PTA).

Case Presentation: 54 years old male patient with past medical history of type 2 diabetes, hyperlipidemia, hypertension, end stage renal disease, peripheral arterial disease with a previous right trans-metatarsal amputation presented to the hospital with left second toe gangrene and underwent amputation of this toe. PTA was done on the left popliteal artery and tibioperoneal trunk. A month later, the patient presented with left foot ulcer with cold feet and excruciating pain. On repeat angiography, it was not possible to cross the distal posterior tibial artery lesion with traditional endovascular techniques. Therefore, arterial venous flow reversal from the posterior tibial artery to posterior tibial vein was performed using a 0.018 CXI support catheter with a 0.014 Astato 30 mg tip wire to puncture the artery and penetrate the vein. Venous filling of lateral plantar and superficial dorsal veins was noted. Three days later, patient underwent left trans-metatarsal amputation and bleeding at the wound site showed adequate perfusion. Four months later, complete wound healing was noted.

Discussion: The greatest challenge to revascularization is in patients with heavily calcified and chronically occluded pedal arteries. This patients lack reasonable distal targets for PTA or bypass surgery.The incidence of major amputation is shown to be significantly high in CLI patients with no intervention (34%/year) as compared with those with intervention (3-5%/year). As desperate times require desperate measures, this has led to the need to explore new treatment options in these challenging subset of patients. Our case illustrates the dramatic wound healing after trans-metatarsal amputation.

Conclusion: Venous arterialization as a viable option should be considered before major amputation is decided for this subgroup of ‘no option’ patients.

Poster #7c

LEFT CIRCUMFLEX GIANT CORONARY ARTERY ANEURYSM WITH INTRALUMINAL THROMBUS

Aditya Hendrani, Nelson Telles-Garcia, Guillermo Sangster, Kalgi Modi
Presenter: Aditya Hendrani MD, LSU Health Shreveport

Introduction: Giant coronary artery aneurysm (GCAA) is a rare coronary artery annomaly defined as aneurysm with diameter greater than quadruple the reference vessel diameter. GCAA with diameter >5 cm is extremely rare with reported prevalence of 0.02%.

Case Presentation: An 84-year-old frail man with history of hypertension, diabetes, persistent atrial fibrillation, and bladder cancer post-chemoradiation was brought from assisted living facility after losing his balance resulting in a fall with minor head trauma without loss of consciousness. Head CT ruled out intracranial bleeding. Chest X-ray revealed an enlarged cardiomediastinal silhouette and multiple retrocardiac well defined opacities with eggshell calcifications (panel A). Echocardiogram showed restrictive diastolic filling pattern, severe pulmonary hypertension, small pericardial effusion, and large coronary artery aneurysm behind posterior wall of left atrium measuring 4.5 x 6.5 cm (panel B-C). Subsequent standard contrast enhanced CT of the chest demonstrated multifocal circumflex coronary arteries ranging in size from 1.0 cm to 7.4 cm in widest dimensions with thrombus formation (Panel D-F).
Further invasive evaluation was deferred because of advanced age, frailty, and severe comorbidities. The patient was treated with anticoagulation and close follow-up.

Discussion: GCAA bears significant risk of acute myocardial infarction and sudden death through thromboembolism, congestive heart failure, fistula formation, compression of surrounding structures (including heart chambers and SVC), aneurysmal rupture with hemopericardium and tamponade. Surgical correction with resection and ligation of aneurysm combined with coronary artery bypass graft is the preferred method based on observational data and case studies. Coronary intervention with covered stent has been reported in GCAA with smaller size.

Conclusion: Due to its rarity, no treatment modality has proven survival benefits. The optimal treatment strategies for GCAA remain unclear and require further investigations.

Poster #7d

LEFT ATRIAL POSTERIOR WALL THROMBUS AFTER POSTERIOR WALL ABLATION

Shourjo Chakravorty, Sangeeta Shah MD, Michael L Bernard MD PhD
Presenter: Shourjo Chakravorty MD, Ochsner Health System

Introduction: Posterior wall isolation for recurrent atrial arrhythmias is a commonly used technique to achieve long-term freedom from atrial fibrillation. Achievement of durable posterior wall isolation has limitations, most notably esophageal heating, and incomplete isolation of the posterior wall resulting in macro reentrant left atrial arrhythmias. Despite widespread use, long-term effects of posterior wall isolation on left atrial function are unknown. Specifically, the effect of isolated atrial walls on stasis and risk of thrombus have not been well established. We present a case of posterior wall thrombus detected 6 weeks after isolation attempt of the posterior wall. This is just the second report of a left atrial posterior wall thrombus in the setting of posterior wall isolation.

Radiofrequency, cryoablation, and surgical ablations have vastly improved the outcomes atrial fibrillation management compared with conventional medical therapy. The type of ablation that is done is decided by the clinician depending on what is best for each patient. Although arrhythmias do recur after the initial ablation attempt, repeat procedures are successful in increasing the overall long-term disease-free success rates. Known causes for recurrence include incomplete pulmonary vein isolation, extra pulmonary vein triggers, and macro reentrant circuits generated from pulmonary vein isolation (PVI). A specific strategy for adjunct ablation after isolation of the pulmonary veins is not well established within the electrophysiology community. One approach to achieve long-term success is to isolate the posterior wall of the left atrium1. This is usually accomplished by linear ablation connecting the superior and inferior portions of the posterior WACA lines (Figure 1). This technique results in a continuous zone of electrical isolation from one set of pulmonary veins, across the posterior wall to the opposite sided pulmonary veins. Though often executed successfully, there is limited literature about the possible complications of using this technique.2

For patients who are refractory to one or more antiarrhythmic agents, or do not want to take such agents, radiofrequency ablation therapy has become the standard of care. Though the success rate for a single PVI for long standing atrial fibrillation is 50-70% with recurrence rates of 20-30%, the procedure is still the best modality for long term treatment for symptomatic patients.13 A common approach for those who fail an initial pulmonary vein isolation procedure is to isolate the posterior left atrial wall during subsequent procedures2-5. This allows for the ablation of the non-pulmonary vein sources that are creating the abnormal rhythm. While indications for when to do a posterior wall isolation varies amongst providers, isolation along the posterior wall carries unique risks relative to the remainder of the left atrium. Most prominently, the posterior wall is in close proximity to the esophagus requiring meticulous attention to esophageal heating. Also, the relative thinness of the posterior wall can increase LA perforation risk. Other known complications of performing a posterior wall isolation include generation of macro reentrant arrhythmias with incomplete linear ablation sets. This case highlights important

considerations for clinicians when evaluating patients post procedure. First, it is important to consider that the formation of a thrombus can be a complication of the actual procedure of isolating the posterior wall. Second, this is an unusual location for a left atrial thrombus which could be missed if the posterior wall is not visualized on imaging studies. Third, operators who perform ablations should be aware of possible risk of forming a posterior wall clot during an ablation which carries an unknown risk of stroke and systemic embolism.

Case Presentation: A 67-year-old female with a past medical history of hypertension, non-ischemic arrhythmia-induced cardiomyopathy with an ejection fraction of 10-15%, persistent atrial fibrillation treated with apixiban 5mg twice a day, and sick sinus syndrome, presented for repeat radiofrequency ablation for an macro reentrant left atrial rhythm. Her electrophysiologic history began with a cryoballoon pulmonary vein isolation. 11 months later, a dual chamber pacemaker was placed for treatment for subsequent tachy-brady syndrome. One month later, she developed recurrence of persistent atrial fibrillation, requiring a radiofrequency ablation procedure. At that procedure, her pulmonary veins were isolated (Figure 2A) and the posterior wall isolation was performed (Figure 2B). Within the next three weeks, she developed a macro reentrant left atrial rhythm with resultant heart failure. Cardioversion and antiarrhythmic therapy were unsuccessful and she was scheduled for her repeat posterior wall ablation 6 weeks after her initial ablation. However, a TEE prior to the procedure revealed a 15 x 11 mm LA thrombus along the posterior wall leading to cancellation of the second posterior wall isolation and adjustment of her oral anticoagulants (Figure 3).Her apixaban was increased to 10 mg bid for three weeks after which a repeat TEE revealed no left atrial thrombus. She subsequently underwent successful ablation of the left atrial, mitral annular, macro reentrant rhythm. The inferior portion of her posterior wall was active with conduction block along the roof. Clinically, the posterior wall was partially isolated. While the the superior (roof) line held up from previous procedures, the inferior line had gaps. (Figure 4 A & B). Post ablation, she had only non-sustained arrhythmias by pacemaker interrogation. She developed no complications of stroke, transient ischemic attack or systemic embolism throughout the entire course of her arrhythmia care and her EF normalized with the restoration of sinus rhythm.

Discussion: The posterior wall of the left atrium provides a substrate for the perpetuation of persistent AF. Electrical isolation of the posterior wall for those with persistent AF after initial RFA remains a management option.1 However, posterior wall isolation may result in the formation of a posterior wall clot in select patients with particular risk factors. This case highlights a possible confluence of factors that may contribute to the formation this unusually located left atrial thrombus. The literature suggests that up to 90% of AF induced clots form in the left atrial appendage.6 Because there is limited literature on this specific complication, we must consider what factors made this patient at a particularly increased risk for this unusually located clot to form. Enlargement of the heart chambers can be caused by a number of conditions. Enlargement of these chambers causes stasis of the blood, and increased risk of clot formation, particularly in the left atrium and left atrial appendage.7-8 The patient in this case has a history of non-ischemic cardiomyopathy, and

moderate mitral regurgitation, two conditions that enlarge the chambers of the heart, both independently and synergistically increasing the risk for clot formation. On paper suggests that chronic mitral regurgitation can cause a rare "Type C" calcification in the posterior wall of the left atrium, increasing the risk of a thrombus in this location specifically.9 The patient is also noted to have mild aortic regurgitation which plays an indirect role contributing to stasis in the left atrium. This patient's history is also significant for acute on chronic heart failure with a left ventricular ejection fraction between 10-15% at the time of the posterior wall thrombus finding. The literature suggests that heart failure, with low ejection fraction is an independent and major risk factor for clot formation.8 The duration of symptomatic AF is also significantly associated with thrombus formation risk. After being symptom free for about three years following an initial RFA, the patient repeatedly complained of irregular heartbeats, palpitations, weakness and fatigue, despite medical and surgical interventions. In a very similar case, a 77 year old women underwent a Maze procedure for management of aortic valve disease and paroxysmal atrial fibrillation, and eventual pacemaker implantation for sick sinus syndrome. Two years' post op, a large thrombus was found in this patient's posterior left atrium as well.10. This chronology of cardiac diagnoses and management is quite similar to that of the patient presented in this case. The formation of left atrial clots is increased after performing RFA due to damage being caused to the wall, especially in patients with pre-existing mitral valve conditions, such as the patient presented in this case.11-12

Conclusion: It is likely that a complex combination of risk factors which includes hypertension, heart failure with low ejection fraction, mitral valve disease, coupled with the recurrence and duration of symptomatic AF, atypical atrial flutter, and worsening heart failure triggered the formation of this posterior wall clot in this patient. Though these cases are rare, especially in those without mitral valve damage (unlike this patient), it is important to recognize that a posterior wall isolation or similar procedure such as the Maze procedure may itself be causing and/or contributing to the formation of a thrombus. It may be worthwhile to perform more frequent echocardiograms to look for such thrombi in the left atrium in similar patients to the one presented in this case to minimize the risk of systemic embolization events in such patients.

Poster #8a

DRUG-RELATED COMPLETE ATRIOVENTRICULAR BLOCKADE CAUSED BY DILTIAZEM

Ryley McPeters MD, Tejas Joshi MD, Micah Mathai MS4, Dr. Jorge Martinez MD, JD
Presenter: Ryley McPeters MD, LSU Health New Orleans

Introduction: Calcium channel blockers (CCB) have been linked to atrioventricular (AV) block.[1] AV block is a delay or interruption in transmission of impulse from the atria to the ventricles due to impairment in the conduction system.[2] Diltiazem is a non-dihydropyridine CCB commonly used to treat various cardiac conditions. Diltiazem has been associated with various types of heart block.[1,3,4] We present a patient (pt) with symptomatic bradycardia with two separate types of AV block secondary to Diltiazem therapy.

Case Presentation: An 80 y/o male with Type 2 DM and HTN on Diltiazem presented with fatigue, chest discomfort & exertional dyspnea. Labs were significant for Hgb of 12.7 & creatinine of 1.5. Troponin, BNP, and chest x-ray were unremarkable. Initial EKG showed 1st degree AV heart block with T-wave inversions & a ventricular rate of 61 BPM. EKG taken 1 hr later showed AV dissociation, 3rd degree complete heart block, & a ventricular rate of 44 BPM. Temporary venous pacer was placed. After ICU monitoring; pt returned to NSR with normal HR & pacer was removed. Pt was discharged & advised to follow up with cardiology for placement of a loop-recorder. During the pt's follow-up, he remained in NSR & 1st degree AV block. Diltiazem was the causative agent for his complete heart block.

Discussion: Diltiazem toxicity can result in heart block in pts on chronic therapy, concomitant chronic renal failure (CRF), elderly pts with CRF due to higher elimination half-lives,[3,5] electrolyte disturbances[6] and with underlying AV conduction disease.[3] 56% of pts whose AV block resolved after discontinuing the offending drug experienced recurrence. True drug-induced AV block occurs in only 15% of pts who have 2nd/3rd-degree AV block. Beta-blockers & digoxin are more likely to cause AV block than Diltiazem.[7]
There are no specific treatment guidelines for heart block induced by AV nodal blockers. Dialysis is ineffective in removing Diltiazem. Research dictates monitoring pts with a transvenous pacer, removing the causative drug, providing supportive care & identifying the underlying conduction disease. Toxicity is extremely rare in chronic Diltiazem therapy with normal electrolytes & hepatorenal function. [6]

Conclusion: Our pt's advanced age & underlying conduction abnormality, as evidenced by his 1st degree heart block on follow up EKG, led to Diltiazem toxicity resulting in a complete heart block.

Poster #8b

DOUBLE DE NOVO RECURRENT SPONTANEOUS CORONARY ARTERY DISSECTION

Lina Ya'qoub, MD, Magdy Hanna, MD, Aditya Hendrani, MD, Cynthia Noguera, MD, Kalgi Modi, MD

Presenter: Lina Ya'qoub MD, LSU Health Shreveport

Introduction: Spontaneous coronary artery dissection (SCAD) is a rare but important cause of Acute Coronary Syndrome (ACS). Recurrent SCAD can be due to extension of SCAD of index vessel in acute phase or can be late or remote (>30 days) de novo dissection of unrelated vessel. The acute phase de novo recurrent SCAD has not been reported. We present a double de novo case of recurrent SCAD involving multiple vessels successfully treated with conservative and revascularization therapy.

Case Presentation: This is a 49-yo female, with medical history of hypertension, ACS with stenting of the Obtuse Marginal (SCAD misdiagnosed as atherosclerotic CAD) in 2016, presented with worsening chest pain for few weeks. Her blood pressure was 202/100 mmHg on arrival. EKG was normal and troponin 0.93. ACS therapy with Heparin, Aspirin, Plavix, Statin, BB and ACEI. Coronary angiogram on admission day 2 revealed patent OM stent and new SCAD of distal RCA. Given TIMI 3 flow and symptom improvement, SCAD of RCA was treated conservatively.

However, patient experienced severe chest pain a few hours later, with troponin 25.50. EKG showed T wave inversions in lead I and AVL. Repeat coronary angiogram on admission day 3 revealed progressive SCAD of distal RCA into PDA and PLV with TIMI III flow and new SCAD of septal perforator and bifurcating diagonal with TIMI 1 flow. IVUS confirmed the dissection and a large intra-mural hematoma in diagonal. Total three stents were deployed to diagonal artery with restoration of TIMI III flow. Patient's symptoms resolved and was discharged on day five.

Discussion: SCAD has emerged as an important cause of ACS in young women. De novo SCAD involving different vascular territories is rare. SCAD continues to be misdiagnosed as atherosclerosis. An accurate diagnosis is key as invasive strategies are reserved for select patients due to lower procedural success. Our case has several unique features. First, it educates us that not all ACS presentations are due to plaque rupture. Second, she had three separate dissection events. Third, these events involved different vascular territories. Fourth, she was successfully treated with both conservative and invasive approaches.

Conclusion: This case highlights the diagnostic and therapeutic challenges associated with recurrent ACS caused by recurrent SCAD in multiple vascular territories.

Poster #8c

EUSTACHIAN VALVE ENDOCARDITIS: TO TEE OR NOT TO TEE?

Hanyuan Shi, MD. Marjorie Bateman, MD. Francesco Simeone, MD, MPH
Presenter: Hanyuan Shi MD, Tulane School of Medicine

Introduction: The Eustachian valve is an embryologic remnant of the inferior vena cava valve that can form as a thickened fold of endocardium in the right atrium in adults. Eustachian valve endocarditis (EVE) is extremely rare and has only been described 30 times in the literature.

Case Presentation: A 45-year-old man with a history of IV drug use presented with one day of shortness of breath. Computed tomography revealed septic emboli to his brain, lungs, kidneys, and spleen. Blood cultures grew methicillin-resistant staphylococcus aureus (MRSA) and candida albicans. A transthoracic echocardiogram demonstrated mild mitral and tricuspid regurgitation. Transesophageal echocardiogram revealed a large pedunculated and poly-lobulated 35 mm x 14 mm eustachian valve vegetation in the right atrium (Figure A, B in four-chamber view and Figure C is the 3-dimensional reconstruction. Red arrow is the Eustachian valve vegetation) along with a 23 mm x 9 mm vegetation on the left coronary cusp of the aortic valve (Figure D showing mid-esophageal aortic valve short-axis view. Red arrow is Eustachian valve vegetation in right atrium and blue arrow is coronary cusp vegetation). Persistent bacteremia prompted initiation of double daptomycin and ceftaroline coverage for MRSA in addition to fluconazole for candida albicans. The patient suffered multiorgan dysfunction secondary to his septic shock, requiring mechanical ventilation, vasopressors, and several sessions of continuous renal replacement therapy. Cardiothoracic surgery was consulted for prosthetic valve replacement. The patient was deemed a poor operative candidate due to the high risk of reseeding and poor prognostic factors including development of cerebral emboli. The patient remains in critical condition in the intensive care unit.

Discussion: The fetal Eustachian valve assists blood flow from the inferior vena cava through the patent foramen ovale into the left atrium. Persistent Eustachian valves are vestigial and rarely can be associated with endocarditis, tumors, and thrombi. There is estimated to be 4.6% prevalence of Eustachian valves in adults with various cardiac diseases.

The causative agent of EVE has been most commonly Staphylococcus aureus, and IV drug use has been most heavily linked to the development. Other conditions such as indwelling catheters, pacemakers, and immunocompromise also have been associated with EVE. It is hypothesized that in IV drug users, there is increased right side expression of matrix molecules capable of binding microorganisms (serum fibronectin and fibrinogen). Structural abnormalities of the valve, abnormal turbulence of blood flow near valve, and virulence factors in bloodstream infections can lead to endocarditis.

The initial diagnostic modality for EVE is transthoracic echocardiogram (TTE). TTE can suggest possible vegetations in any of the four chambers of the heart but can occasionally

miss the Eustachian valve. Transesophageal echocardiogram (TEE) can view the Eustachian valve best with midesophageal four chamber and midesophageal bicaval views. While the initial TTE in our case only showed trace mitral regurgitation and tricuspid regurgitation, multi-planar TEE revealed very large vegetations on two valves. It is possible that these vegetations grew in size throughout the interval of time between the initial TTE and TEE. Studies have shown that TEE, however, should be performed more liberally with a low threshold for suspected clinical endocarditis, especially in IV drug users.

Conclusion: Our case highlights diagnosis and treatment of a large Eustachian valve vegetation with a concomitant coronary cusp aortic valve vegetation previously not described in the literature. This case also adds to the clinical repertoire suggesting the need of a TEE over a TTE for definitive diagnosis of Eustachian valve endocarditis.

Poster #8d

HYPERTROPHIC CARDIOMYOPATHY MASQUERADING AS CRITICAL AORTIC STENOSIS IN A PATIENT WITH BICUSPID AORTIC VALVE

Avaneesh Jakkoju, MD, Mehnaz Rahman, MD, Murtuza Ali, MD
Presenter: Avaneesh Jakkoju MD, LSU Health New Orleans

Introduction: We present the case of a 59 year old female with undiagnosed but coexisting congenital heart diseases, hypertrophic obstructive cardiomyopathy (HOCM) and bicuspid aortic valve (BAV). She complained of light headedness and progressive NYHA III symptoms to her primary care team and workup including a transthoracic echo (TTE) at an outside facility was significant for BAV with elevated gradients across the left ventricular outflow tract (LVOT) and aortic valve (peak gradient 100 mmHg; mean gradient 60 mmHg). Based on these findings she was referred to Cardiology Clinic for suspected severe aortic stenosis.

Case Presentation: Upon initial presentation to our clinic, her physical exam was significant for borderline low blood pressure around 90/75 mm Hg, an early peaking systolic murmur without respirophasic changes, and preserved S2 heart sound. Her TTE was reviewed; it was discovered that she had increased intra-cardiac velocities, a significantly thickened LV with hyper dynamic function and cavity obliteration. Her aortic valve itself had excellent leaflet mobility. Asymmetric septal hypertrophy >3cm causing dynamic outflow obstruction was also noted and bicuspid aortic valvular anatomy with preserved leaflet mobility was confirmed. Using the continuity equation, her aortic valve area was determined to be 1.8 cm2. With Valsalva maneuver, her LVOT velocity was observed to increase from 1.5 m/s to 4m/s. A diagnosis of hypertrophic cardiomyopathy (HCM) with intracavitary dynamic obstruction with concomitant bicuspid aortic valve without aortic stenosis was made.

Discussion: HCM is an autosomal dominant disease caused by a family of genes encoding cardiac sarcomeric protein components. About 2/3 of HCM patients will also have LVOT obstruction and are labeled as HOCM. BAV is the most common adult congenital heart disease with natural progression to valvular stenosis in a bimodal age distribution. The prevalence of BAV among the HCM is reported at 0.9%, which parallels the prevalence in the general population. Timely recognition of the primary obstructive process in patients with concurrent disease is essential. Misdiagnosis can lead to unwarranted surgery, worsening heart failure and high risk for sudden cardiac death.

TTE is an appropriate initial test especially as the gradients estimated by continuous wave (CW) Doppler echocardiogram shows a strong correlation with invasive measurements. CW measures the peak blood acceleration along its cursor line; the LVOT and aortic valve are too close to distinguish abnormal peak gradient of each. Given the high intra-cardiac velocities, pulse-wave Doppler could have been used to sequentially interrogate from the LV apex down to the LVOT in order to confirm the anatomical level of obstruction. Additionally, for this patient, the small cavity with high cardiac output and preserved EF were evidence against severe valvular aortic stenosis.

Conclusion: Although the prevalence of BAV in HOCM population is low, the cardiac interventions that each necessitate vary widely. Thorough diagnostic evaluation and an understanding of their hemodynamic interplay will be paramount in guiding therapy.

Poster #9a

LIMITATIONS OF ECHOCARDIOGRAPHY IN EVALUATING RIGHT ATRIAL THROMBUS

Ala Mohsen, Avaneesh Jakkoju and Neeraj Jain
Presenter: Ala Mohsen MD, LSU Health New Orleans

Introduction: Thrombus is the most common intra-cardiac mass seen in day to day clinical practice. The left atrial appendage and left ventricular apex LV are two of the most common loci for intra-cardiac thrombus, especially in the setting of atrial arrhythmias and LV dysfunction, respectively. Whereas right sided thrombus is less commonly detected.

Case Presentation: A 64 year old man with past history of known coronary artery disease, heart failure with reduced ejection fraction, hypertension, and interstitial lung disease with chronic respiratory failure presents to the hospital with worsening lower extremity edema. Echocardiography showed normal left ventricular (LV) ejection fraction, moderate right atrial (RA) enlargement and severely enlarged and hypokinetic right ventricle (RV). A computerized tomographic scan to assess for pulmonary embolism (PE) was negative and right heart enlargement was confirmed. Of note was the finding of a low attenuating filling defect in the right atrium anteriorly and superiorly concerning for thrombus. A repeat echocardiogram was performed to visualize the mass but failed to show any right atrial lesion. A cardiac magnetic resonance imaging (MRI) was performed to further characterize the mass which was confirmed as a large lobulated thrombus, sessile within the right atrial appendage (RAA). The mass measured 4.8 x 1.6 x 2.9 cm.

Discussion: This case highlights the limitation of routine transthoracic echocardiography in visualizing certain right heart structures especially RA appendage. Right sided thrombus is less commonly detected as left sided thrombus, it can be seen with a deep vein thrombus in transit or severe RV dysfunction. The RAA cannot be visualized on transthoracic echocardiography routinely due to its anterior and superior location and overlying aerated lung. When suspected, cardiac MRI is the gold standard for diagnosis due to high resolution imaging and ferromagnetic properties of thrombus. Accepted treatment includes anticoagulation for 3 to 6 months as untreated right atrial thrombus can lead to pulmonary embolism. In a patient as ours with pre-existing RV dysfunction, substantial PE could be devastating with severe hemodynamic compromise.

Conclusion: Transthoracic echocardiography is an excellent diagnostic modality with some limitations like visualizing certain right heart structures. High index of suspicion should prompt alternate imaging modality such as cardiac MRI as highlighted in our case.

Poster #9b

MECHANICAL CIRCULATORY SUPPORT IN THE SETTING OF CARDIOGENIC SHOCK FROM TAKOTSUBO CARDIOMYOPATHY

Tariq Yousuf, MD; Paul Katigbak, MD; Ahmad Jabbar, MD; Charisse Ward, MD
Presenter: Tariq Yousuf MD, Tulane School of Medicine

Introduction: The presentation of Takotsubo stress cardiomyopathy (TSC) is variable. Contemporary observations reveal the rates of cardiogenic shock, need for mechanical circulatory support and death may have been previously underestimated. Patients with suspected Takotsubo Cardiomyopathy in the setting of cardiogenic shock have been shown to benefit from additional mechanical support from Impella when conventional medical therapy is not effective. We present a case of TSC with cardiogenic shock refractory to inotropes necessitating hemodynamic support.

Case Presentation: A 69-year-old female with a medical history of hypertension, obstructive sleep apnea and depression presented to the emergency department with shortness of breath. She was found to have chest pain, evidence of volume overload and respiratory failure due to pulmonary edema on initial evaluation. Her ECG showed non diagnostic ST-T changes. She had pulseless electrical activity (PEA) shortly after arrival to the emergency department and was intubated and resuscitated. Echocardiogram revealed an ejection fraction of 20-25% with severe global hypokinesis. CT angiography of the chest was negative for a pulmonary embolism. She underwent emergent coronary angiography which revealed nonobstructive coronary artery disease. Right heart catheterization revealed severely elevated filling pressures (pulmonary capillary wedge pressure of 40) and a cardiac index (0.9 L/min/m2). Hemodynamics did not improve despite aggressive diuresis and increasing doses of inotropic support. Impella CP (Abiomed, MA) was successfully placed with acute improvement of clinical and hemodynamic parameters. Her left ventricular ejection fraction improved within 24 hours, and the impella was removed the following evening. Upon discharge from the hospital, echocardiogram revealed a completely normalized ejection fraction of 65%.

Discussion: There have been no randomized control clinical trials demonstrating the benefit of Impella in patients with cardiogenic shock in the setting of Takotsubo Cardiomyopathy, but there are several published case reports documenting the usefulness of an Impella in this clinical scenario. A case report revealed benefit of Impella CP in a patient with TSC and refractory cardiogenic shock1.

Conclusion: The diagnosis of cardiogenic shock warrants increased awareness for early recognition of symptoms and subsequent initiation of treatment. As our patient was clinically deteriorating despite conventional treatment of cardiogenic shock, an Impella CP was implanted that eventually lead to complete recovery of ejection fraction on subsequent echocardiogram.

Poster #9c

PREGNANCY MANAGEMENT IN A PATIENT WITH CONGENITAL HEART DISEASE WITH SEVERE RV TO PA CONDUIT STENOSIS AND REGURGITATION

Sajan S Gill BS, Kelly K Shum MD, and Sangeeta B Shah MD
Presenter: Sajan Gill BS, Ochsner Health System

Introduction: Pregnancy results in multiple hemodynamic changes that places a significant stress on the cardiovascular system. With the advancements in medical and surgical care, many individuals with complex congenital heart disease are now living into childbearing years. Much still needs to be understood about the effects and best management of pregnancy in an individual with complex congenital heart disease.

Case Presentation: We describe the management and successful delivery of a 27 year old pregnant female with repaired Tetralogy of Fallot or ventricular septal defect with pulmonary atresia. Our patient presented with New York Heart Association (NYHA) Class II symptoms and was found to have severe pulmonary conduit stenosis with a peak gradient of >75 mmHg.

Discussion: Management of a pregnant patient with adult congenital heart disease (ACHD) should initially involve risk stratification for complications (commonly congestive heart failure exacerbation and arrhythmias) using established tools such as the modified WHO Pregnancy risk score. Based upon the patient's risk category, consideration should be given for the frequency of patient follow up, anesthesia options, and mode of delivery. Patients in moderate to high risk stratification should ideally be managed by a multidisciplinary team at a specialty center, and all patients should undergo an anesthesia consultation prior to delivery. Decision for vaginal or cesarean delivery should be made on a case by case basis with consideration given to requests of the mother. Patients with asymptomatic moderate to severe pulmonic stenosis can be managed conservatively with appropriate follow up and cardiac imaging, allowing intervention to be completed at a later date.

Conclusion: With appropriate follow up and specialist care, a patient with moderate-severe pulmonic stenosis with unrestricted pulmonic insufficiency can be managed with a conservative approach to ensure a safe and successful delivery.

Poster #9d

PREGNANT WITH INFECTIVE ENDOCARDITIS OF UNKNOWN ETIOLOGY

Rebecca B. Lee, DO, Stephen D. Anderson, MD, R. Clark Cutrer MD, Nathalie S. Malcolm, MD, Brandon W. Lennep, MD, Elizabeth A. Meyer, MD, Hannah Copeland, MD, Rachael Morris, MD, R. Patrick Cochran, MD, Myrna E. Alexander-Nickens, MD
Presenter: Rebecca Lee DO, LSU Health New Orleans

Introduction: While infective endocarditis (IE) itself is a common occurrence, IE in the presence of pregnancy is exceedingly rare. It often has catastrophic consequences for both mother and child. Here we report a non-fatal case of methicillin sensitive staphylococcus aureus (MSSA) IE in a primigravida 20 week gestation.

Case Presentation: A twenty-three-year-old caucasian primigravida at twenty weeks gestation, without significant past medical history, presented to an outside hospital complaining of severe posterior flank pain of three days duration. Pain was associated with fever, chills and sore index finger. Transesophageal echocardiogram (TEE) showed a 1.2 x 1 cm mass on the aortic valve (AV), consistent with endocarditis. The patient was started on IV nafcillin and transferred to The University of Mississippi Medical Center (UMMC). On arrival, the patient was tachypneic and tachycardic. A loud III/VI diastolic murmur noted at the base of the heart. A tender, red nodule on the distal phalanx of the right index finger as well as splinter hemorrhages on the feet were also noted. Transthoracic echocardiogram (TTE) showed mild aortic insufficiency and a mobile mass on the ventricular aspect of the AV. Patient denied intravenous (IV) drug use. Urine drug screen was negative. Once the patient was stabilized, it was determined that she could be managed in the outpatient setting with IV nafcillin. Seventeen days after being discharged, she returned to the OSH with hemoptysis, chest pain and shortness of breath. TTE revealed worsening aortic insufficiency and left heart failure. She was transferred back to UMMC. The patient was started on lasix for pulmonary edema and hydralazine for afterload reduction. IV nafcillin was continued for infection control. A multi-disciplinary approach was utilized to prolong the pregnancy and allow for optimal fetal development. The patient was delivered by cesarian section at 28 weeks. Shortly after delivery, the patient decompensated and required immediate valve replacement. After valve replacement, she stabilized and was successfully weaned from all supportive therapy. The patient was discharged home 14 days later. At the time of delivery, the infant was hypotensive requiring fluid resuscitation and dopamine as well as a course of hydrocortisone. An echocardiogram on day 15 of life showed patent foramen ovale and a moderate patent ductus arteriosus (PDA). The infant was in the neonatal intensive care unit until she was discharged home on day 68 of life.

Discussion: IE in pregnancy is exceedingly rare. Early diagnosis and management of IE are essential to achieving better outcomes in pregnant women. In the patient presented here, a multi-disciplinary team approach and appropriate medical management were utilized. This allowed the fetus to reach a viable gestational age prior to delivery which ultimately lead to the survival of both mother and child.

Conclusion: Patients presenting with new onset heart murmurs, vascular phenomena such as splinter hemorrhages, immunologic phenomena such as Osler's nodes and infectious symptoms should be worked up for IE.

Poster #10a

PREMATURE CORONARY ARTERY DISEASE IN SETTING OF PROBABLE FAMILIAL HYPERCHOLESTEROLEMIA

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Presenter: Christen Harris MD,

Introduction: Premature Coronary Artery Disease is defined clinically as coronary artery disease (CAD) before age 45 in males and 55 in females. Although, lipid abnormalities are a risk factor for CAD at any age, specific lipid patterns and clinical presentations are suggestive of patients who may be at higher risk for premature CAD. Familial Hypercholesterolemia (FH) is an autosomal dominant disease with marked LDL and cholesterol elevations that frequently result in premature CAD. Here, we present a case of a 26 year old man diagnosed with premature 3-Vessel CAD in the setting of probable FH.

Case Presentation: A 26 year old man with no significant medical history presents to the hospital after onset of intermittent, substernal chest pain lasting minutes while at rest. Similar pain and palpitations were present for the preceding three months associated with physical exertion at work and relieved with rest. Patient denies smoking or illicit drug use and is not obese. Family history significant for maternal sister with premature CAD. Vitals on presentation are significant for a BP of 210/115 at presentation. Physical exam and bedside echocardiogram are unremarkable. Lab values on presentation are significant for elevated cholesterol 332 mg/dL, LDL 276 mg/dL, HDL 37 mg/dL, and Triglycerides 79 mg/dL. EKG demonstrates NSR without abnormalities concerning for ischemia. Initial troponin is elevated at 0.45 ng/mL and peaks at 3.12 ng/mL. A diagnosis of NSTEMI is made which prompts further assessment with coronary angiography revealing 3 vessel CAD requiring subsequent CABG.

Discussion: This is a rare case of severe premature 3-vessel CAD. Clinical suspicion for FH should arise in such cases with characteristic lipid abnormalities. FH is caused by the presence of genetic mutations resulting in impaired LDL catabolism. Heterozygous FH is typically seen during adulthood (> age 20). In the absence of definitive genetic testing, clinical criteria can assist in the diagnosis. This patient has a Dutch Criteria score of 8 which indicates probable heterozygous FH.

Conclusion: Due to the severe cardiovascular effects associated with Familiar Hypercholesterolemia, recognizing and initiating appropriate interventions (LDL- lowering therapy) in probable or definite disease is important for improving prognosis and prevention of devastating cardiovascular events.

Poster #10b

RARE CAUSE OF STEMI IN PROSTHETIC VALVE ENDOCARDITIS

Shashitha Gavini D.O, Farheen Shaikh M.D, Agostino Ingraldi M.D FSCAI, Jody Rosson M.D.
Presenter: Shashitha Gavini D.O., LSU Health New Orleans

Introduction: Prosthetic valve endocarditis (PVE) is a condition with high risk of morbidity due to associated complications such as sepsis, perivalvular abscess, and peripheral embolism. We present here a case of left main coronary artery embolism due to PVE.

Case Presentation: 35-year-old male with history of bioprosthetic aortic valve 8 months ago secondary to infective endocarditis presented with 1 day history of fever and malaise. Vitals on admission with tachycardia 111 beats/min, temp 101.8 °F and 3/6 systolic murmur at aortic area with janeway lesions present on physical exam. Labs significant for lactic acid 2.8, troponin of 4.5 mg/dL and WBC 16. ECG with sinus tachycardia, premature atrial complexes, and right bundle branch block. Patient initiated on empiric antibiotics with blood cultures positive for methicillin sensitive staph aureus (MSSA). Transesophageal echo revealed vegetation on undersurface of bioprosthetic aortic valve with edema versus annular root abscess. Cardiothoracic surgery with evental plans for surgical repair, however on the following day, ECG revealed ST elevations in II, III, aVF, V1-V3 and troponin of 9.7ng/mL. Patient taken emergently for percutaneous coronary intervention (PCI) where the LAD had proximal embolic occlusion treated with drug-eluting stent and ejection fraction 20% with placement of intra-aortic balloon pump (IABP). A presumptive diagnosis of MSSA PVE with embolic myocardial infarction from vegetation was established. Patient became a poor surgical candidate with significant multisystem organ failure and soon decompensated secondary to his overall critical illness.

Discussion: Staphylococcus aureus is the leading culprit in PVE and requires early surgical intervention when associated with aortic root abscess which can result in conduction abnormalities and embolic phenomenon presenting as a STEMI1. PCI and IABP provided temporary stability however, revision of aortic valve replacement was indicated as early as possible.

Conclusion: Embolization from veggitations is an extremely rare phenomenon. It is reported that most emboli to the LAD arise from mitral valve vegetations, making this an especially unusual case2. Early surgical intervention is vital in PVE associated with aortic root abscess as embolic events can become fatal complications.

Poster #10c

REVERSIBLE CARDIOMYOPATHY AND CRITICAL HYPOPHOSPHATEMIA: A CAUTIONARY TALE OF OVER THE COUNTER DIURETICS

John Lee Spitzer MD, PhD
Presenter: John Spitzer MD, Tulane School of Medicine Internal Medicine

Introduction: Critical hypophosphatemia has been associated with reversible cardiomyopathy and arrhythmias.

Case Presentation: A 46 year old woman with a history of prolonged QT, and over the counter diuretic use presented to the ED complaining of nausea and vomiting. Her triage labs showed a phosphorus level of .9, an anion gap of18, and a metabolic acidosis with a carbon dioxide of 18. She was noted to be tachypneic by both the ED physician and Nurse. She was started on PO and IV phos repletion, but was unable to tolerate the PO. Her EKG showed a HR 46 with QTc of 537, which decreased to 505, and then 467 on serial studies. She received Promethazine and Ondasetron, and scopolamine before admission to medicine. She then went into ventricular tachycardia/fibrillation arrest with return of spontaneous circulation after 4 rounds of CPR. EKGs were concerning for STEMI. A bedside echo showed anteroseptal akinesis with hypokinesis in other LV segments; EF was 55%.

Discussion: This patient's hypophosphatemia was likely secondary to salicylate toxicity from an over the counter diuretic that she had been frequently been taking. Salicylate toxicity produces an anion-gap metabolic acidosis and subsequent respiratory alkalosis, which can result in an intracellular shift of phosphorus, as it likely did in this patient. Unfortunately, while she was noted to be tachypneic, there was no arterial blood gas to performed; there was likewise no salicylate screen performed as it was unknown that the diuretic she had been frequently taking was magnesium salicylate.

Conclusion: This case demonstrates the potentially lethal side effect of an over the counter medication as well as the adding to the body of evidence suggesting that critical hypophosphatemia results in a reversible cardiomyopathy.

Poster #10d

THE USE OF TORSEMIDE IN THE ERA OF HIGH VALUE CARE

Neal Dixit
Presenter: Neal Dixit BS, Tulane School of Medicine

Introduction: Nearly 10% of patients with heart failure are readmitted within 30 days. The average admission for heart failure exacerbation costs nearly $15,000. Strategic medication choice can reduce heart failure readmissions, increasing healthcare value.

Case Presentation: Mr. Z is a 70 y.o. man with a history of heart failure, CAD, HTN, and diabetes who presented with shortness of breath for the past 2 days. He recently stopped taking his evening dose of furosemide. This was the 5th time he had been admitted in the last 12 months. ROS was negative. Vitals were stable and within normal limits. Physical exam demonstrated significant volume overload. Laboratory studies showed an elevated creatinine and a BNP of 1100. The patient was diuresed with high dose IV furosemide for an acute on chronic heart failure exacerbation. Within a few days, Mr. Z's condition improved. Over several conversations with Mr. Z, he opened up about his difficulties with medication adherence. He said it was difficult to remember to take his evening doses because "he is always on the go" and forgets to take his medications with him throughout the day. He also complained about waking up at night to urinate after taking his evening diuretic dose, commenting that when he felt alright, he skipped it so he could sleep better. Considering this, we sought to adjust his medication regimen to ensure that all were taken in the morning. Furosemide was his only medication dosed twice daily. We opted for once daily dosing of torsemide- due to longer half-life, instead. Mr. Z was very pleased with the changes to his regimen. After further counseling, he was discharged.

Discussion: Heart failure exacerbation is a leading cause of 30-day readmission. Studies have shown that lack of medication adherence is associated with readmission. Studies have also shown that twice daily dosing of medications is associated with lower adherence than once daily dosing. Torsemide is a loop diuretic with a longer half-life than the more commonly used furosemide. Additionally, torsemide has been shown to have several other advantages over furosemide.

Conclusion: With the recent emphasis on High Value Care and penalties for hospital readmissions, torsemide could be the preferred loop diuretic of choice in patients with systolic heart failure.

Poster #11a

USE OF A PERIPHERAL STENT IN A LARGE RCA THROMBOTIC LESION

Paul Katigbak MD, Tariq Yousef MD, Rohit Maini MD, Charisse Ward MD, MPH
Presenter: Paul Katigbak MD, Tulane School of Medicine

Introduction: Large culprit diameters were predictors of worse prognosis in patients presenting with STEMI undergoing primary PCI (1). We report a STEMI case which angioplasty was done using a balloon expandable peripheral stent on a large diameter lesion.

Case Presentation: A year 50 year old man was brought in by EMS after complaints of mid sternal crushing chest pain while at a gym. EKG on the field showed junctional bradycardia and ST segment elevations in the inferior leads. En route, he was afebrile and hemodynamically stable. He was loaded with ASA and ticagrelor.
In the cath lab, he received heparin for anticoagulation. A 6 French JR4 guide catheter (Medtronic, Minneapolis, MN USA) was advanced over a wire and positioned at the RCA ostium. There was a 99% occluded proximal RCA with large thrombus burden and TIMI-0 flow. A Pro-water wire (ASAHI Intecc, Tustin, CA USA) was advanced across the culprit lesion. Balloon angioplasty was performed using a 2.5 x 15 Apex balloon catheter (Boston Scientific, Marlborough, MA USA) at the mid to proximal RCA. A transvenous pacemaker was placed due to bradycardia. A PRONTO extraction catheter (Teleflex Inc. Morrisville NC, USA) was used followed by balloon angioplasty with a 4.0 x 12 Apex balloon catheter (Boston Scientific, Marlborough, MA USA). IVUS (Volcano Corp., San Diego, CA USA) was used to determine the size of the vessel. A 5.0 x 18 followed by a 6.0 x 18 and a 6.0 x 15 Herculink BMS (Abbott Lab, Abbot Park, Ill USA) was deployed at the mid to proximal RCA with small overlap of the previously placed stent. Angiography showed good stent expansion with TIMI-3 flow. The door to balloon time was 44 minutes. Post PCI, transvenous pacing was weaned and EKG showed resolution of his ST segments and he remained chest pain free. At 6 month follow up he is doing well.

Discussion: There has been only one case report with use of a balloon expandable peripheral stent to treat a culprit aneurysmal SVG graft (2). To our knowledge, there have not been any publications of the use of a peripheral stent on a large native coronary artery during a presentation of a STEMI.

Conclusion: Patients presenting with ST elevation myocardial infarction with a large diameter culprit lesion can be safely treated with balloon-expandable peripheral stent to match the large vessel size.

1. Pedersen F et al. JACC. 2014. 64(20).2101-2108.
2. Picard F et al. Journal of Medical Cases. 2016. 7 (4):133-135.

Poster #11b

UTILITY OF RECOGNIZING EARLY EKG CHANGES IN A SUBTYPE OF TAKOTSUBO CARDIOMYOPATHY IN THE SETTING OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE EXACERBATION: A CASE REPORT

Maria Khan, Husam Watti, Khagendra Dahal
Presenter: Maria Khan DO, LSU Health Shreveport Internal Medicine

Introduction: Takotsubo cardiomyopathy is characterized by reversible systolic dysfunction of the left ventricle in the absence of any significant obstructive coronary artery disease. Most commonly, the apical and the middle segments of the left ventricle are seen to be akinetic. Its clinical presentation is said to be indistinguishable from myocardial infarct. The gold standard used to differentiate ACS from TCM is coronary angiography as it clearly shows if a significant obstructive lesion is present. This is a unique case presentation as it not only illustrates an uncommon subtype of Takotsubo cardiomyopathy (TCM), but also very clearly exemplifies specific EKG changes meant to aid in distinguishing TCM from Acute coronary syndrome (ACS). This was previously thought to be indistinguishable on the basis of electrocardiogram (EKG).

Case Presentation: A 63 year-old Caucasian female with past medical history of chronic obstructive pulmonary disease (on 2 liters home oxygen), coronary artery disease (status post RCA stent in 2000's) and anxiety who presented to the emergency department for acute exacerbation of COPD. The patient had been experiencing 2 weeks of worsening shortness of breath, productive cough and increased sputum production. She had been using her inhalers more frequently at home and had increased her home oxygen to 3 liters, still with only minimal relief. On the day of presentation to the emergency department, the patient called EMS as she was concerned about her breathing.

When the patient arrived in the emergency department, her Physical exam revealed a blood pressure of 98/74, pulse 101, temperature 97.5 °F (36.4 °C), temperature source Oral, respiratory rate of 27, and oxygen saturation of 95 % on 3-4 liters of oxygen. Scoliosis of spine was also noted. Her electrocardiogram (EKG) showed sinus tachycardia with ST elevations in leads V3-V6 as well as low voltage QRS complexes when compared to previous EKG from 12 weeks prior (Figure 1). Initial troponin on presentation was 2.74. Cardiology was consulted for evaluation. The patient was advised to have an emergent left heart catheterization (LHC). However, the patient refused initially and wanted only medical management. She was not a candidate for thrombolysis due to history of SAH. Her troponin peaked at 5.16. A 2-D echocardiogram was done which showed mid and distal septal, anterior, lateral and apical wall akinesis concerning for ischemia in the left anterior descending (LAD) territory. The patient was then agreeable to have the LHC 3 days after the initial presentation. LHC showed patent right coronary artery (RCA) stent with only mild luminal irregularities noted in the RCA. Left main was angiographically normal, as was the left circumflex (LCx). Mild to moderate stenotic lesions were noted in LAD, Diagonal 1 and Ramus Intermedius. A Left ventriculogram revealed hyperkinetic basal segments and akinesis of the apex and periapical segments consistent with Takotsubo cardiomyopathy.

The patient was treated for chronic obstructive pulmonary disease (COPD) exacerbation.

Her breathing improved, and she was back to her baseline 2 liters oxygen use. She was discharged to a long term acute care facility with cardioprudent medications ASA 81mg, Atorvastatin 80mg, carvedilol 12.5mg BID and losartan 25mg. Her inhalers included tiotropium, fluticasone-salmeterol and ipratropium-albuterol. The patient was also started on sertraline 50mg and instructed to continue her home medication of lorazepam 0.5mg every 6 hours as needed for anxiety. Follow up Echo and EKG were planned; however, patient had passed away due to worsening of the pulmonary disease.

Discussion: The Mayo Clinic diagnostic criteria, proposes that 4 criteria must be met in order to diagnose TCM: these criteria are, transient hypokinesis, akinesis or dyskinesis of the left ventricular mid segments, new ECG changes mimicking acute MI, absence of angiographic evidence of obstructive coronary disease, and absence of pheochromocytoma and myocarditis.

It has been widely suggested that a bronchogenic sub type of TCM exist. Although not as common, there have been some reported cases of TCM in the setting of COPD or asthma. Some authors even estimate that pulmonary pathologies are suspected to be found in as high as 44% of TCM cases, thus justifying the creation of a separate classification as a bronchogenic subtype. The disproportionate predominance of sympathetic activity during a COPD exacerbation has been noted as a possible trigger for TCM. Rajwani et al described 5 specific cases of bronchogenic TCM and noted some distinguishing features. The absence of chest pain, as with our patient, is noted as a main distinguishing feature of the bronchogenic subgroup of TCM. Progressive dyspnea was also a commonality noted in all 5 cases; this was also present in our patient.

TCM is commonly seen in postmenopausal women, as with our patient. Also in the setting of TCM, wall motion abnormalities are seen to extend beyond the distribution pattern of any single epicardial coronary artery, as seen in our case report. Though TCM is thought to be indistinguishable from ACS on the basis of EKG findings, Madias et al described a noticeably low voltage and attenuated QRS complexes in association with TCM seen in over 90% of the EKGs reviewed for published literature of TCM cases. Since then, many other cases have been reported showing marked QRS complex attenuation in the setting of TCM. In our case, a previous EKG from 12 weeks before the patient presentation was available for comparison (Figure 1). Low QRS Voltage, which is defined as ≤5 mm in limb leads and/or ≤10 mm in precordial leads, and significant attenuation of QRS complexes was appreciated. The likely mechanisms of this phenomenon was explained by author Madias who explored myocardial edema versus counterbalancing of depolarization vectors as the likely mechanisms and concluded that myocardial edema is the most likely cause of the low QRS voltage appreciated in EKGs of TCM patients. This was supported by review of multiple TCM cases where patients had cardiac MRI performed that revealed myocardial edema. Madias used Ohm's law (Voltage= Current x Resistance) to explain how changes in the resistance of the electric conductor caused by myocardial edema can account for this phenomenon of low QRS voltage (LQRSV) and attenuation of the amplitude of the QRS complex (AAQRS) since anything that alters resistance or current will result in changes in voltage. Extracardaic pathologies such as pulmonary congestion and pleural effusion, would also influence these changes.

Conclusion: TCM is a transient reversible systolic dysfunction and is estimated to be the culprit in 1-2% of patient presenting with clinical symptoms of a ACS. These patients do not require treatment with heparin drip and P2Y12 inhibitors thus it is important for clinicians to have a high level of suspicion to identify the correct etiology. As suggested by author Madias, utilizing EKG changes can aid in early identification of TCM and differentiation of TCM from ACS.